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(President's Address)
NEW MEXICO STATE MEDICAL SOCIETY

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(Delivered to the forty-ninth annual meeting, held at Albuquerque, N. M., May 20 to 22, 1931.)

While we have made great progress in medicine, there are some things that change not. From time immemorial it has been the custom at medical meetings for the President's Address, instead of discussing medical progress, to depart from that field and give a philosophical dissertation on some phase of medical economics. It is assumed that the president of a medical society is a man of broad experience and mature judgment and therefore his address takes a course somewhat between that of fatherly advice and a philosophical sermon.

State medicine and medical legislation are two subjects that are discussed freely in presidents' addresses. State medicine is the big bogey that is staring us in the face at the present time and I presume that before we are through we will have it in some form. They have tried it in several countries and a great many people who cannot distinguish change from progress, feel that we should try it in this country. In the time allotted me, I could not begin to pay my respects to state medicine, so I will pass that by.

Medical legislation is another subject that has been worn threadbare. I wonder if we do not make a mistake in bothering with medical legislation at all, for we are misunderstood; when we endeavor to protect the public unselfishly, they think we are actuated only by selfish motives. The mentality of the average member of the legislature is such that he cannot conceive of our being actuated by anything but selfish motives, when we are really trying to protect the uninitiated.

The task of educating legislators is surely a Herculean one and were we successful in educating the legislators and getting them to realize the importance of protecting the public to the point that human lives would not be turned over to the care of people whose time spent in the preparation for their profession is less than the time required to train a good barber, we would still have the governors to work on and the average governor is of the type somewhat like they say of the Harvard man,—"You can tell a Harvard man anywhere, but you can't tell him much." I believe as far as legislation is concerned, we would do better to go ahead, put our own house in order, do our work honestly and conscientiously and wait patiently until an enlightened public demands some protection themselves.

I believe, however, that we owe a great deal to the cults and should perhaps let them alone for they do two things for us, by their activities: they stimulate us to better work and once in a while, they do discover a grain of wheat in many bushels of chaff and regular medicine does now and always has, gladly, and I might say, anxiously, appropriated any beneficial measure that has been brought out from any source. Homeopathy taught us to modify the enormous doses that were in vogue a hundred years ago; the electric taught us to give more attention to the relief of symptoms: the osteopaths, while working on a false premise, developed some methods that have been adopted by the physiotherapists and the christian scientists have emphasized the cultivation of optimism, hopefulness and cheerfulness. So, I have chosen to talk today, more upon some of the things they do not teach in medical schools. Do not misunderstand me and think I am

finding fault with our medical schools. The progress that has been made in medical education in the last generation is almost inconceivable, however, I wonder if we are not getting to be almost too scientific, or I would not say too scientific, but, in becoming ultra-scientific, we are becoming less practical. It is an open secret that there is constant warfare in every medical faculty today, between the laboratory man and the practical man, with the laboratory man winning at every turn. Naturally, the laboratory men, all being full-time men, devote their entire thought to the teaching work; the practical men, many of them, besides teaching some of the things they know and use in their every day work, make their living down at the office and thus do not have the time for thinking and planning for the school and curriculum.

One professor of medicine, who had held that chair in three of our great universities, wanted his students to be able to spend a little more time than they were in the wards, had the temerity to insist that he be given these students for more time to take them through the wards and let them see some sick humans, but the laboratory men insisted on keeping them in the laboratory stimulating frogs, experimenting with serpents, insects and chickens when, he argued, they might be studying human beings, somewhat as Lowell put Cromwell's soliloquy when contemplating flight to the Colonies or fight with King Charles I. He says:

> "And what should we do in the small Colony of pinched fanatics who would rather choose, Freedom to clip an inch more from their hair, than the great chance of setting England free?"

This professor, of whom I speak, quit teaching and engaged in private practice and he has since told some of his friends, he never knew there was so much money in the world as he is able to make since he quit teaching.

Thus, medicine lost a great teacher and his boys will become a rich man's sons instead of the sons of a poor professor. I mention this because it is more or less typical of what is going on in the various universities, as members of the teaching force in different schools have openly admitted our schools teach a great deal of anatomy, physiology, chemistry, biology, pathology, history, diagnosis and treatment of disease, stressing all the while the disease. This is all well and good, but should we not also teach psychology and devote time to the study of human nature? For our success or failure rests more largely on our ability to understand and manage our patient than any other one thing and, that ability to understand people is the turning point in any business or profession. It is said of Rufus Choate, one of the greatest lawyers of his day, that he could talk to a jury and by watching closely their facial expressions, tell when he had won them to his views and, it might be said of any physician who has become great, that he did so because he understood people.

Are we losing sight of the Patient? We study disease; we think disease; we think in terms of pathology and forget that the greatest variable in the whole question is the patient. What we are concerned with, is not so much the disease, but the reaction of the disease on this particular patient and we all react in a different manner. For instance, you can take a solution of some foreign protein and inject one drop into the skin of four different individuals. In one you will get no reaction at all; in another you will get a slight reddening of the skin; in the third you will get a fair sized wheal and perhaps in the fourth you may get a large bleb, a general urticaria, an attack of asthma, a gastro-intestinal upset, a severe allergic reaction. Our mental reactions vary just as much as our physical. You may go down the street, engage in an argument with four different individuals. In the course of argument with each one, use that short and ugly word "liar". One of these men, placing no value on your opinion whatever, will not be ruffled in the least and simply grin; a second will retort you are another, and a third will knock you down and give you a good thrashing and a fourth will carry a grudge and resolve at the first opportunity to do you "dirt." The individuality of the individual is the thing we must consider.

What then does a patient want and what motives actuate him to consult a physician? It may be that he is suffering pain and it always is that he is worried. He wants relief from some thing that is bothering him. He is discouraged. What does he expect when he consults a physician and what does he want to get? It seems to me the first thing he wants to do is tell you his story, tell you what is worrying him, tell you what troubles him and, as Sir William Osler said, "If you let your patient talk long enough, he will tell you both what the trouble is and what he thinks will cure it." At least he wants to tell his story to a sympathetic listener. Besides that, he wants a study of his condition, sufficiently searching that he feels you have gone far enough into his case to find out what it is that is bothering him. After he has gone that far and has told you the things which seem to be troubling him and you have examined him and made a

searching study of his case and he feels you now have enough information to understand him and to know what the trouble is, he wants to hear from you. It may be he does not care for any treatment; he may simply be worried; he may be afraid, as a great many people are, that he is developing a cancer, (since the lay magazines are discussing so much the danger of cancer we have a great many mental cancers to contend with) and he may want to be assured, if there is any possible way of doing so, that he has no cancer or that he has no other insidious condition developing of which he need be afraid. Each year more and more people are coming in for a careful examination and what they want is to be assured that, so far, there has been no break in the machinery. If your patient is suffering pain, he wants something to relieve that pain, but more than anything else and more than everything else he wants encouragement. I think most of our patients come to us on account of being discouraged about something than any other one thing and I do believe that we, as medical men, should give each patient a careful examination, listen to his story and find out as near as humanly possible what is the trouble with him, direct and advise him, but above all else we should encourage him.

We should think in terms of the patient and not in terms of pathology and disease, for after all, our entire thought and study is for the benefit of the patient. But, many times we become so enthused over our work that we lose sight of our objective, as a man being shown through a new, modern teaching hospital and was shown this for the benefit of the nurses, that for the benefit of the students, this for the benefit of the doctors, that for the benefit of the visitors and when they got all through, he innocently asked "and what for the benefit of the patients?" In their anxiety to benefit every one, they had lost sight of the very ones for which the hospital had been built.

So let us keep ever before us the patient. Let us study him carefully, think of his needs, his troubles, his worries; take a sympathetic interest in the whole problem; encourage and comfort him and if we do that we will have nothing to fear from medical legislation or state medicine. We will enjoy our work and will find that it gives us not wealth, but as President Elliott has put a fine phrase, it does give "The durable satisfactions of Life."

THE SPECIFIC TREATMENT OF PULMONARY TUBERCULOSIS

JOHN W. FLINN, M.D.,
Prescott, Arizona

(Read before the Fortieth Annual Meeting of the Arizona State Medical Association, held at Nogales, May 7-9, 1931.)

The ultimate object of medical science is the prevention of desase. Until this can be accomplished, the profession must needs devote a large amount of time and attention to its second most important objective—the discovery of a specific cure for each individual disease.

Specific has been defined as "a remedy having a definite curative action in relation to a particular disease or symptom, as genuine in relation to malaria." In this sense the use of the remedy is not necessarily limited to the disease or symptom in question. Quinine has a beneficial effect in other diseases than malaria, although in no other are its effects so striking and so uniform.

The word specific has a second and more limited significance when we refer to the specificity "manifested in the relation . . . of an antibody to its antigen, or the reverse." We use the word in this more limited sense when we speak of diphtheria antitoxin as a specific cure for diphtheria. In this class of cases the use of the remedy is confined to the one disease.

In this paper the word specific will be used in the former and broader sense of a remedy having a definite curative effect in tuberculosis, but whose use is not limited solely to the treatment of that disease.

In the past half century a stupendous amount of work has been done in the field of tuberculosis. In the whole history of medicine no other disease has been studied so extensively and intensively. So striking and important have been the discoveries by laboratory workers regarding the nature and action of the tubercle bacillus and the reaction of the body tissues to infection with this organism, that they have very largely overshadowed the quieter, less spectacular work of hundreds of clinicians, in every civilized country, on the treatment of the disease tuberculosis. So much is this so that the rank and file of the medical profession have apparently not yet begun to realize that in the past twenty years a remedy has been discovered which is as specific in its effects in pulmonary tuberculosis as is quinine in malaria, and which is almost, if not quite, as effective as is antitoxin in diphtheria. If the medical profession will use this remedy as it now uses antitoxin in diphtheria, the in tuberculosis as early and in as heroic doses

results will be just as striking and almost, if not quite, as favorable.

Before proceeding to discuss this remedy in detail, let us remind ourselves that even diphtheria antitoxin will exert its full beneficial effect only in the very early stages of the disease. Leading authorities state:

"Early administration of antitoxin is urgently advocated in any case where there there is membrane in the throat and where there is a suspicion of diphtheria. With eace day's delay in giving antitoxin, the danger from diphtheria increases alarmingly. The earlier the remedy is administered, the more certain and rapid is the effect. In cases of any severity where diphtheria is suspected, and in cases of croup, it is far better to administer the remedy at once, making a culture at the same time, than to delay the treatment until a diagnosis has been made by bactero'ogic examination. The first injection should be large enough to control the disease. The intravenous method of injection is used entirely for severe and septic cases. Laryngeal diphtheria causes the greatest number of deaths. Sixty per cent of the cases admitted as croup require intubation and intubated cases under three years do very poorly; over three years the percentage of recovery is 70 to 80."

THE SPECIFIC REMEDY

It is now about twenty years since rest in bed began to be advocated as a valuable remedy in pulmonary tuberculosis. At that time our ideas regarding its indications were very vague; our knowledge of its dosage was extremely limited. After a quarter of a century of careful, painstaking observations in large numbers of cases, we are just beginning to realize how really specific this remedy is in its actions, and when and in what doses it should be used.

Nearly thirty years' experience in the use of rest in bed in the treatment of all forms of tuberculosis has convinced the writer that it will effect a clinical cure in an extremely large percentage of all cases of pulmonary tuberculosis, if administrated in the early incipient stage and in large doses. It will **not** cure large numbers of cases of widely-spread miliary tuberculosis; but when the disease has become definitely localized in the lungs, rest in bed, if used early, will effect a clinical cure in a very high percentage of all cases.

Not only so, but this same remedy will produce a clinical cure in a majority of cases of moderately advanced pulmonary tuberculosis, if properly administered for some considerable time. Even in far advanced cases with marked cavitation, rest in bed will effect permanent improvement and a return to a moderately useful life, in quite a large number of cases, if used in large doses and over long periods of time. Has antitoxin proved any more effective in diphtheria?

ADMINISTRATION

When should rest in bed be used in pulmonary tuberculosis? The initial dose of rest in bed should be administered just as early in pulmonary tuberculosis as the first dose of antitoxin is given in diphtheria. In other words, when the symptoms are strongly suspicious, do not wait for a positive diagnosis. Administer your remedy at once and make your definite diagnosis later. Put your patient on rest in bed under careful observation until the diagnosis can be definitely confirmed. So far as the death rate is concerned, pulmonary tuberculosis is a much more deadly disease than diphtheria. If the physician who fails to administer antitoxin early in diphtheria is guilty of gross carelessness, the one who does not give rest in bed early in pulmonary tuberculosis should be convicted of criminal negligence.

DOSAGE

In what dosage should rest in bed be used in pulmonary tuberculosis? Unfortunately, the word used to designate this remedy is one which has been in use in common conversation in the English language for several centuries. For this reason it is most difficult to limit its significance in a specific scientific sense. Consequently we have found it necessary to qualify the significance of even the phrase "rest in bed" by the use of adjectives.

As soon as even a tentative diagnosis of pulmonary tuberculosis is made, every case should be immediately placed on, at least, "fairly complete rest in bed." This means that the patient is in bed more than twenty-three hours a day, being allowed up only for bathroom privileges. He has all meals in bed and has bed-baths. If, at the end of a week, his highest temperature is not below 100 degrees, or if he has a persistent, disturbing cough, he should be put on "complete rest in bed," which means twenty-four hours in bed every day, with use of wash-basin, bed-pan and urinal.

If at the end of a month there has not been some improvement in the condition of the lungs and in the blood picture, even if there has been a noticeable improvement in the general condition of the patient and in his symptoms, he should be put on "absolute rest in bed," which entails the constant attendance of a special nurse, who feeds the patient and prevents any unnecessary movement in bed, even in the use of the sputum cup.

Note well that in any phase of this regime it is not enough that the patient be in bed, he must rest in bed. Even in fairly complete rest in bed, where the patient is allowed bathroom privileges, he should lie flat in bed relaxed for at least one hour in the middle of the forenoon and should have at least two hours of complete relaxation, lying flat in bed and if possible asleep, in the middle of the afternoon. Nothing should be permitted to interfere with the patient's rest. If the cough disturbs, he should be given codeine. If his digestion is impaired, his diet should be carefully regulated, and, if necessary, vitamins or digestive medicines administered. If he is restless by day or sleepless by night, bromides or some other hypnotic should be given for short periods. Encouragement and other forms of suggestion are very valuable means of inducing rest.

DURATION

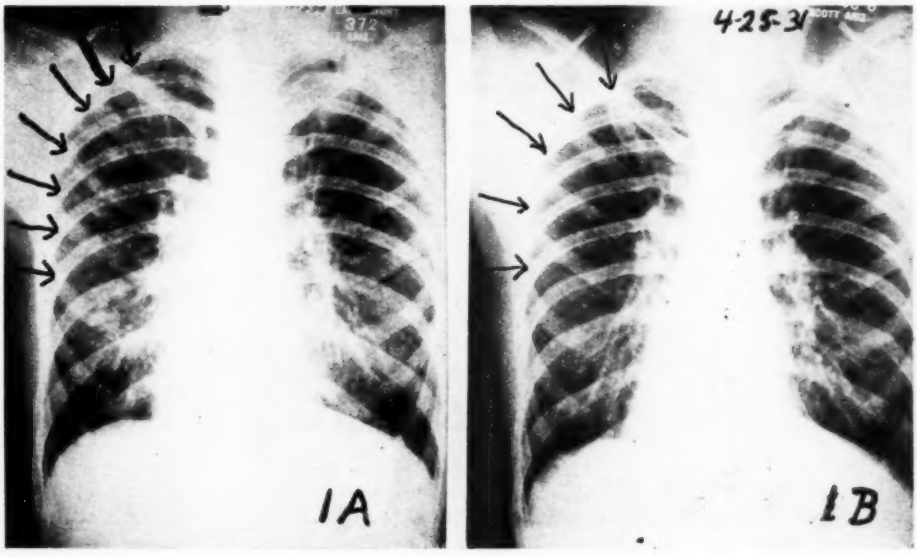
Over how long a period should rest in bed be continued and in what dosage? When the proper dose of rest in bed has been determined in each individual case, as outlined in a previous paragraph, that dose should be continued until repeated, careful examinations show neither symptoms nor signs of any active disease in the lungs, and until the scar tissue is hardened to a considerable extent. It is not enough to have the patient free from all symptoms—elevation of temperature or pulse, cough, expectoration, loss of weight, and so forth. In addition, physical examination, stereoscopic x-ray films and blood picture must all show definitely that all lesions in the lungs are healed over and that the scar tissue interlacing and surrounding the lesion is no longer soft and permeable. Then, and then only, should inincreasingly sma'ler doses of rest in bed be prescribed. If, after several weeks of carefully graduated partial rest in bed, careful examination discloses neither symptoms nor signs of active disease and shows thicker and harder scar tissue, very carefully graduated walking exercises should be prescribed.

PREVENTION

Not only is rest in bed, when properly administered, a specific remedy in the treatment of pulmonary tuberculosis, but it is an invaluable aid in the prevention of this disease. If rest in bed in proper dosage could be administered to every case of active, pulmonary tuberculosis, there would be very few cases of far-advanced pulmonary tuberculosis at the end of a generation, thus eliminating, to a very large extent, massive infection in childhood. One-third of a century later the mortality from tuberculosis would be insignificant and its morbidity in adults very small.

CONCLUSIONS

The solution of the problem of pulmonary tuberculosis in man is very largely in the hands of the general practitioner—the man engaged in the general practice of medicine. It is to him the patient comes when this disease is in its incipiency and it is by him the treatment can and must be administered, if the ravages of the disease are to be quickly controlled. The research workers and clinicians who have been limiting their work to the study and treatment of pulmonary tuberculosis, have discovered a specific remedy for this disease. The remedy and the



technic of its administration are easily within the reach of every physician. Responsibility for the clinical cure and the ultimate prevention of pulmonary tuberculosis now rests squarely on the shoulders of the medical profession. All that remains is for physicians to apply it properly.

ILLUSTRATIVE CASES

Case 1. Male, age 22 years. Had a suspicious illness at 14 and was sent to the mountains for six months at 18. Active tuberculosis of lungs accidentally discovered in March, 1921. Very few symptoms. Physician advised sanatorium treatment. Instead, patient spent a year and a half living very quietly in summer and winter resorts of California. Gained 20 pounds in weight and felt fine. Examination in September, 1930, showed spread of disease. Referred to us October 1, 1930.

Lung condition October 2, 1930. (Figure I. A.): Left lung; infiltration with fuzzy edges from apex to upper border of 7th rib and 10th V. S. Area suspicious of cavitation beneath the clavicle; right lung, infiltration from apex to first interspace, fuzzy edges.

Put on "fairly complete bed-rest" (23 hours a day in bed, meals in bed and bed-baths; up only for bathroom privileges). In a few days all symptoms disappeared. On same routine until May 1, 1931. Seven months in all.

Lung condition April 25, 1931 (Figure 1. B). Left lung area suspicious of cavitation in left apex cannot be seen; infiltration areas discrete and hard. Right lung, infiltration areas mostly discrete.

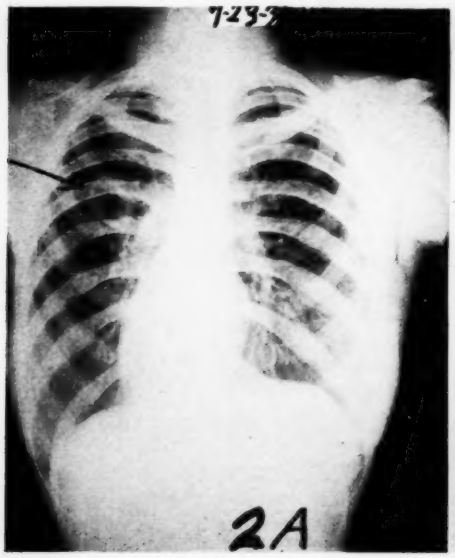
Now getting rest in bed in constantly decreasing, carefully graduated doses, that is he is being allowed to sit up for increasing periods each day on a very carefully graduated schedule.

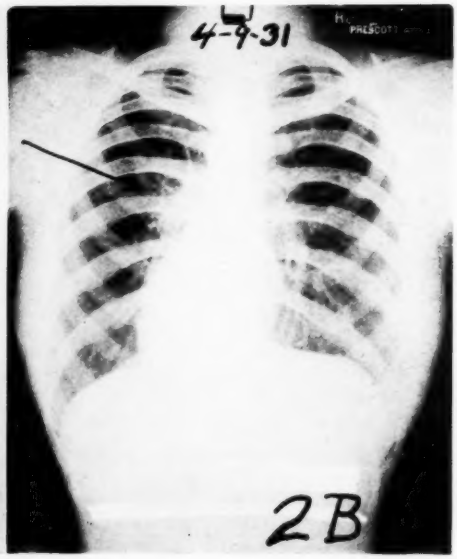
COMMENT

In our opinion the lesions in this patient's lungs are now healed over and the scar tissue is no longer soft and permeable. If his exercise is carefully controlled for several months, the scar tissue will grow thicker and harder and he will very likely make a good clinical cure. What a pity he had not taken his physician's advice and gone to bed eighteen months before he did! He wasted at least two years of his life, to say nothing of the risk he ran of losing his chance of a good recovery. Better still, if this lung had been given proper rest in bed and subsequent care when he was sent to the mountains at eighteen, it would undoubtedly have prevented his present prolonged illness.

Case No. 2. Female, aged 26 years. History of repeated colds for nearly ten years, but general health fairly good until April, 1930, when she developed persistent cough. Continued to work hard until a definite diagnosis of active pulmonary tuberculosis was made June 25, 1930. Entered sanatorium July 3, 1930, complaining of severe cough, dyspnea, general soreness in left chest and excessive fatigue. Sputum abundant and containing tubercle bacilli. The x-ray films taken at that time have been lost, but physical examination showed an active lesion in the left lung extending from apex to 4th rib, with a shower of fine moist rales in the second interspace just above what seems to be a small cavity lying outside of the hilum on this side. numerous fine and medium moist rales after cough in left interscapular space and some pleural rales in left base. An old healed lesion in the upper part of right lung.

She was placed at once on "fairly complete rest in bed" and responded very promptly. By latter part of September 1930, the cough had completely cleared and there was practically no sputum. At that time stereo x-ray films showed (Figure 2A) an area of decreased density suggestive of cavitation in the second interspace on the left side immediately in front of the seventh vertebral spine, measuring 3x2 cm. not surrounded by a very firm wall. Reaching out from the hilum into the second, third and fourth interspaces the linear markings are increased with small areas of fuzzy densities along them..

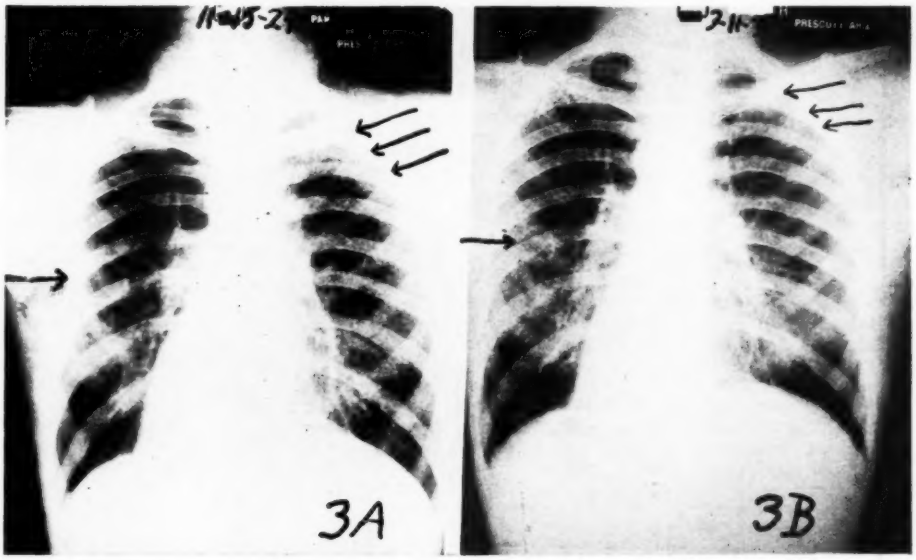




Examinat'on on November 5th showed very little change. Only a slight increase in the amount of scar tissue. At that time much against our better judgment we were induced to allow this patient to sit up for very slowly increasing periods each day. She made indifferent progress until early in January, 1931, when she had a very slight hemorrhage. We placed her at once on absolute rest in bed under the constant care of a special nurse. She again responded very promptly. In a very few weeks all symptoms had again completely disappeared. On April 1931, x-ray films (Figure 2B) showed that the area suggestive of cavitation in the upper left lung had quite cleared up, and that the scar tissue had begun to appear hard.

COMMENT

This patient will remain on complete bed rest for another month or two and then will be placed on partial rest in bed and later on very carefully graduated exercise. She should make a good clinical cure. What a pity we had not known enough to put her on absolute rest in bed when she first entered the sanatorium. It would have saved her several months of very valuable time. Better still, if she had had proper rest in bed ten years ago, when she first developed repeated colds, or even in April, 1930, (instead of June, 1930) what an amount of time and expense could have been saved!



Case 3. Male, 28 years. Health began to fail in 1925, six years ago. Chills, fever, sweats; very little cough; much aching; marked pleurisy left side. A few months later glands on both sides of neck swelled and developed a skin lesion. Traveled all across the continent and was treated for all manner of diseases, including maralia and hyperthyroidism. Cervical glands broke down; were opened and discharged for 18 months.

Three years later (in 1928) diagnosis of tuberculosis was made at the Crile Clinic and sanatorium treatment advised. Home physician disagreed. Operated on glands on both sides of neck. Used stock vaccine and autogenous vaccine from patient's sputum.

In 1929 came to Arizona and diagnosed chronic malaria and sent to hot springs and later to dude ranch. In August, 1929, examined at Mayo Clinic and again diagnosed tuberculosis. Entered sanatorium October 1929.

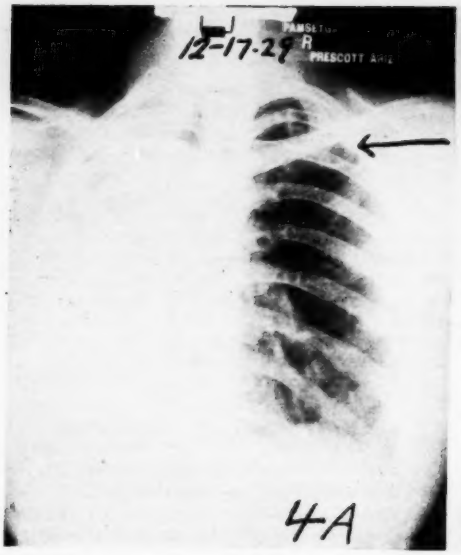
Films exposed at Mayo Clinic, August 9, 1929, showed tuberculosis of the right upper lobe with two cavities and tuberculous involvement of the upper left lobe with a single cavity. Films exposed November 15, 1929, (Figure 3A) showed a cavity in the right lung in the first interspace, one interspace in diameter, and cavity of same size in the second interspace. In the left lung a cavity 3 cm. in diameter in the posterior axillary line from the upper border of the seventh rib to the lower border of the eighth.

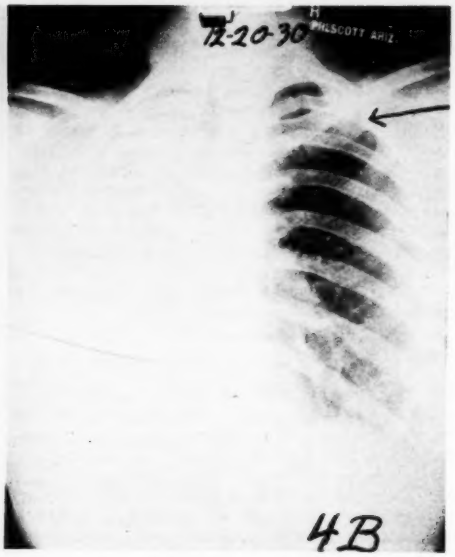
Patient was placed on "fairly complete rest in bed" with a careful diet and very carefully graduated sun baths to all the body except the chest. He soon began to improve very slowly.

In December, 1930, that is after 14 months' treatment, the glandular condition was completely healed over. X-ray films exposed March 11, 1913 (Figure 3B). Right lung, the two cavities in the right apex have entirely disappeared and cannot be seen; left lung, lying immediately beneath the eighth vertebral spine in the posterior axillary line is a well defined cavity 2 cm. in diameter. It is smaller by 1¼ cm. in diameter measurement than on Nov. 15, 1929.

COMMENT

This patient will be continued on his same schedule of rest in bed and partial sun baths. The probabilities are that the left lung also will heal completely without artificial compression. What a pity this patient did not accept the advice given in 1928 and get "rest in bed" treatment. Better still, what a pity a definite diagnosis was not made in 1925 and "rest in bed" administer-





ed properly! This would have saved him at least five or six years of disability and expense.

Case 4. Female, age 29 years. Active tuberculosis first diagnosed in 1928. In bed two months. Afterwards up and around but in poor health. Entered sanatorium July, 1929. At that time there was soft infiltration in the right lung from apex to the second rib with a cavity shadow smaller than a dime in the second interspace. It was difficult to interpret the x-ray films of the left lung, but physical signs strongly indicated extensive multilocular cavitation. This patient was placed on "fairly complete rest in bed" and soon began to improve slowly. At the end of six months (December, 1929) (Figure 4A) definite cavitation had taken place in two areas in the upper part of the right lung; one immediately behind the first rib (2 cm.) and another immediately beneath it, in the first interspace (3 cm.). Outside of these two areas, there had been a marked clearing up throughout the upper part of the right lung, the infiltration having become clear-cut and discrete. There seemed to be some clearing in the left lung which was less opaque, especially over base.

A year later (December, 1920) (Figure 4B) the cavities in the upper right lung had disappeared. They appear to be contracted to the small area of increased density now lying immediately beneath the first rib and clavicle and third vertebral spine. The edges of the infiltration in the upper right have become clear-cut and discrete. There was no essential change in the left lung.

COMMENT

This patient will continue on "fairly complete bed-rest" for a few months more until the scar tissue around the former areas of cavitation in the upper right becomes thicker and harder. Then some form of artificial compression will be used on the left side. We will propably attempt an artificial pneumothorax. If that is not successful we will consider phrenic neurectomy. The probabilities are that an extensive thorocoplasty will be necessary to completely heal the left lung. What a pity prolonged rest in bed had not been used in 1926 when she was first taken ill!

DISCUSSION

DR. SAMUEL H. WATSON, Tucson. (opening discussion): Whenever we have Dr. Flinn on our program, we are assured of a good paper. This paper of his was not only good, but it was a most unusual presentation of one of the cost important problems in medicine today.

I dare say that not a man in this room has even thought of "bed rest" as being a **specific** in the cure of pulmonary tuberculosis, and yet, as Dr. Flinn has so plainly told you, it is just that, properly used. It is about time we had such a paper to jolt us out of our lethargy of absolutely unjustified self satisfaction. I am inclined to think that too many of us who are treating this disease have been "lulled to sleep," so to speak, about the seriousness of it because of the reports we read each year of the constantly diminishing death rate. Twenty-five years ago we were told that in any city of the United States of one hundred thousand population we had 220 deaths from tuberculosis. Now for the same sized city we have decidedly less than half of that number, about ninety in round numbers. We say a drop from 220 to 90 is some accomplishment in a quarter of a century in a disease so serious that it was at that time counted the most serious of all chronic diseases in its fatal results. We called it then the "Captain of the Men of Death." We don't call it that any more. We hear that it has fallen to fifth or sixth place as a cause of death, that heart disease, pneumonia, cancer and so forth, far outstrip it now. But I want to call your attention to the fact that most of our decrease in mortality from tuberculosis in the past twenty-five years, has not occurred in pulmonary tuberculosis, nor in the important age period when this disease mostly occurs. Please, don't forget that in the period of twenty to forty years of age, pulmonary tuberculosis is still the "Captain of the Men of Death," and causes one out of every four or five deaths that occur. If this be true, and I assure you that it is true, if pulmonary tuberculosis is the most curable serious disease in the world today, as

some of us know it to be, if we have such a simple specific cure as Dr. Flinn has suggested, then there is something wrong with the doctors that treat this disease.

I want to say now that I agree with every word Dr. Flinn has to say in his paper except his suggestion for the solution of the problem. Dr. Flinn said "The solution of the problem of pulmonary tuberculosis is largely in the hands of the general practitioner." I want to speak in defense of the general practitioner in this connection, in fact, I want to compliment the general practitioner for the advance he has made in the handling of his tuberculosis cases over the last twenty years. He is making constantly earlier diagnoses in his pulmonary cases, and taking better care of those he treats. But he is constantly treating less and less pulmonary tuberculosis, he is turning more and more of them over for treatment to the tuberculosis specialist. It is to the latter and not the former we must turn for the solution of this problem. He is mainly to blame because he does not use bed rest as Dr. Flinn has told you it must be used to cure this disease. Talk to the average chest specialist, ask him what he thinks is the most important thing in the treatment of this disease, and his answer will invariably be—rest, but ask him for details as to how he carries out his rest program and you will usually find that his patients are loafing, or at the best spending most but not all (every minute) of their day in bed, seldom indeed bed resting as completely as they should be, unless the patient happens to be so sick he prefers to stay in or cannot get out of bed. To sum up then: here we have the most curable chronic serious disease in the world today. We have a specific cure "bed rest" but the mortality is still appalling. How are we to overcome this? My answer is: by insisting that the tuberculosis specialist learn what rest really means and accomplishes in this disease, then get him to use it and to cure his patients will be easy. If every such specialist could be made to start using BED REST tomorrow as Dr. Flinn uses it, the solution of the problem of pulmonary tuberculosis would soon be accomplished.

DR. CHAS. BROWN, (San Diego): Stated that many of the cases seen by them suffer greatly from laryngeal tuberculosis. They have found that these can be relieved by the application of trichloracetic acid directly to the lesions.

DR. F. F. MILLER (Miami): Stated, in substance, that their great difficulty was in securing the cooperation of patients for prolonged periods of rest. Most of the cases of tuberculosis observed by them are in families of workers and it is economically difficult or impossible for them to go to bed for long periods of time. While not taking issue with the correctness of Dr. Flinn's statements, it is not always easy to carry out ideal rest programs.

DR. JOHN W. FLINN (closing). I had no thought of unkindly criticism of the man in general practice. To me he is the backbone of the medical profession and occupies the most important and most honorable position in our profession. My thought was that pulmonary tuberculosis is such a widespread disease that it is quite impossible, for a tithe of the cases, even, to have the services of a specialist. Therefore the general practitioner must needs acquire expert knowledge of pulmonary tuberculosis, and it is to the tuberculosis specialist that he should look for expert advice. To me it has been a very great satisfaction to have a physician of Dr. Watson's standing concur so fully regarding the importance of rest in bed in the treatment of pulmonary tuberculosis.

In replying to Dr. Miller, I have no remedy to acid has a good effect in a certain number of cases of laryngeal tuberculosis is because it relieves pain and gives the patient both local and general rest.

In repllying to Dr. Miller, I have no remedy to offer for the patient who is not financially able to take the rest treatment. This is, of course, the tragedy of the situation. But conditions in this respect are certainly improving from year to year.

It is not, however, hard to make a patient realize the seriousness of his condition, if the physician is rather brutally frank with him at the first examination. In my opinion it is criminal not to be absolutely frank. The very life of the patient depends on his being told that unless he follows directions implicitly he cannot and will not recover his health. With especially obdurate patients it is much better to tell them frankly that they will surely die in a comparatively short time if they do not give themselves the intensive care outlined. Absolute frankness with the patient is the key to the situation. Unless you are very frank with the patient, you cannot gain and retain his confidence. Without the patient's full confidence and cooperation it is extremely difficult to treat pulmonary tuberculosis successfully.

A STUDY OF INTESTINAL TUBERCULOSIS

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(Read before the fortieth annual meeting of the Arizona State Medical Association, held in Nogales, Arizona, May 6th to 8, 1931.)

Ancient and modern medicine alike have recognized the occurrence of intestinal tuberculosis as a complication in pulmonary tuberculosis and, until recently, alike regarded its manifestations as precursors of the fatal termination of the disease. In recent years, however, due to a method making possible the earlier diagnosis of the disease, modern medicine, as in other fields, has been enabled to assume an aggressiveness toward this complication which has reduced its status from that of the incurable disease to that of a serious disease frequently cured and, except in the near terminal type, practically always susceptible of improvement.

Credit for this changed attitude should be given Stierlin, whose observations concerning the changed physiology of the colon in the presence of ulceration were the first given to the medical world. Also to Pierie who, acting upon the suggestion of Archibald, examined and reported on several patients referred to him because of suspected intestinal tuberculosis. His observations led him to practically the same conclusions reached by Stierlin. Also to Stewart, pupil of Brown and Sampson, who has frequently and ably given the medical world the results of his studies and researches. Perhaps most credit is due Brown and Sampson, whose work has placed medical practice in relation to intestinal suberculosis upon a basis as to diagnosis and treatment compar-

able with other chronic and serious diseases long treated with fair success.

Due, no doubt, to the hopeless outlook in this disease for centuries, the medical profession has been slow to acquaint itself with the principles and technic of the early diagnosis of intestinal ulceration, as evidenced by the answers received in answer to a questionnaire sent to over a score of recognized leaders in the tuberculosis field in America. Answers received from those acquainted with and using this method were enthusiastic as to their belief in its accuracy and the results of treatment.

TECHNIC

The technic employed in these studies was modeled after the Brown-Sampson technic which has for its purpose an x-ray examination of the gastro-intestional tract by aid of the barium meal at such hours as makes possible the study of the cecum and colon. Fluoroscopic examination is made at the sixth and seventh hours after the meal and as often thereafter as the specific circumstances of the case make necessary. X-ray films are made at the sixth, seventh, eighth and twenty-fifth hour periods following the administration of the meals, or at such other hours as are necessary to obtain the needed information. Rarely are examinations at later hours necessary.

PREPARATION OF PATIENTS

Patients are to have no laxative forty-eight hours prior to the examination. No breakfast is permitted the morning of the examination. Eating is resumed at the noon hour.

THE TEST MEALS

The primary test meal consists of

Barium sulphate	oz. IV
Cocoa	oz. I
Sugar	oz. I
Flour	oz. I

mixed and stirred well in sixteen oz. of sweet milk and given to the patient a three o'clock on the morning of the examination.

A secondary meal composed of a mixture of

Barium sulphate	oz. II
Buttermilk	oz. XVI

is given at the sixth hour examination (immediately after the sixth hour film in made) to make possible the gastric examination and an observation of the amount and location of the opaque substance at the twenty-fifth hour. (9 a. m. the following day.)

This time and manner of the meal administrations meet the requirements of the examination. The patients do not object. They make possible later examinations, during the working hours of the x-ray staff, of those patients in whom there is delayed filling of the cecum and colon.

X-RAY EXAMINATION

The first film is taken at the sixth hour (prior to the secondary meal administration.) This records the state of the stomach as regards a residue. The patient is then immediately fluoroscoped and given the secondary meal. The fluoroscopic observations of the stomach are made in the usual manner at this examination.

The opaque enema was used as a means of possible check upon cases presenting x-ray evidence of disease. In only one study, however, did the enema give more conclusive information than the meal. This case came to autopsy and our positive findings were verified.

The seventh hour after the primary meal a film is made and the patient fluoroscoped. At the eighth hour a film only is made unless special indications for fluoroscopy are present.

Fluoroscopy is done at the twenty-fifth hour after the secondary meal in order that structures may be identified and a film is made for record.

INTERPRETATION OF FLUOROSCOPIC EXAMINATION AND X-RAY FILMS

Notes supplemented by graphic drawings if necessary should be made at the time of the fluoroscopic examinations, noting:

1. Location of cecum.
2. Amount of filling in the colonic tract.
3. Motility of the cecum under palpation.
4. Areas of tenderness elicited by palpation.
5. Size and shape of cecum.
6. Condition of haustra, i.e., their regularity, whether deep or shallow, and the condition of filling.
7. Constancy of appearance at later examination.
8. Head of column.
9. Abnormalities of movement along the colonic tract.

The following should be noted (when the film is interpreted).

1. The size and shape of the cecum is described. Note: The normal cecum is the broadest part of the colon.
2. The amount of filling of the cecum as compared with the adjacent ascending colon is noted. Note: The irritable cecum may not contain as much opaque material, as the colon immediately distal to it.
3. The character and regularity of the haustra are investigated. Note: The normal cecum presents regular and fairly deep haustra. The irritable

cecum (also ascending colon) may be narrowed, have shallow haustra, missing haustra, constrictions or irregular indentations or may fail to retain barium in many quantity.

4. The retention of barium by the cecum is noted. Note: the normal cecum retains barium longer than any other portion of the intestinal tract. Any empty cecum at the twenty-fifth hour, with barium elsewhere in the colon is strongly suggestive of irritability of the cecum.
5. The retention of barium by the colon is noted. Note: the normal colon retains barium forty-eight hours or longer. An empty colon or a colon with little barium at the twenty-fifth hour is a positive indication of hypermotility.

BASIS OF X-RAY DIAGNOSIS OF CECAL AND COLONIC ULCERATION

The diagnosis of cecal and colonic ulceration is based upon evidence of **local irritability** and, when present, general colonic hypermotility.

The indications of local irritability are:

1. Spasm. Note: Since the cecum and ascending colon are more often the site of ulceration special reference is now had to these portions of the colon. In spasm of the cecum and ascending colon delayed filling of the cecum and a dilated terminal ileum are often observed.

2. Filling defects. Note: Filling defects are indicated by:

(a) A narrowed cecum or ascending colon. (b) Irregular, shallow or missing haustra. (c) A failure of the cecum or the ascending colon to retain barium with evidence of barium both cephalad and distal to these structures. (d) Failure of the cecum to retain a normal residue of barium in twenty-four hours.

Cecal spasm or other occlusions, partial or complete, are often clearly demonstrated by aid of the opaque enema and fluoroscopy. The progress of the head of the enema column should be carefully observed and all hesitancies in its progress noted. Normally there is a delay in passing the flexures but the progress of the enema material through the open colon should be consistent. Often structures under mild spasm will relax under the pressure of the enema and fill very well. It is, therefore, of great importance to observe the filling of the colon and cecum under the fluoroscopic screen since a well filled colon as shown by the film may give information which in truth is inaccurate.

Autopsy findings indicate that when spasm is demonstrated by aid of the opaque meal, and there is failure of demonstration of spasm by aid of the opaque enema, the meal findings should be accepted as most accurate. However, if spasm is demonstrated by aid of the opaque enema, and it is not so clearly shown by the meal, due consideration should be given the enema evidence and further study made. The value of this conclusion was demonstrated when one of the cases came to autopsy, in which the diagnosis (positive) was based upon the enema evidence rather than the meal. (Autopsy findings substantiated our diagnosis).

GENERAL COLONIC HYPERMOTILITY

General colonic hypermotility is indicated by failure of the colon to retain barium for a period within normal limits. The rate of movement, no doubt, is variable in different subjects. However, studies have indicated that at the twenty-fifth hour a large amount of the meal is normally present in the colon. If the colon is empty or retains only a small amount of the opaque substance hypermotolity is certainly demonstrated. (Correct standards for the estimation of normal and abnormal residue of opaque material at any stated period can only be obtained after many observations of colons devoid of movement abnormalities.)

Follows a statistical summary of the work upon which the above observations are based:

Three hundred patients were examined, thirty-five of whom had no clinical tuberculosis. Of the two hundred and sixty-five tuberculous examined, thirty-two were far advanced, with the pulmonary form and had class "A" symptoms. Of this number positive evidence of colonic ulceration was found in two—an incidence of 7.8 per cent. Hypermotility (colonic) was found in both. The enema substantiated the meal findings in both. Neither had symptoms referable to the gastro-intestinal tract. Gastric residue was present at the sixth hour after the meal ingestion in one. Both had pulmonary cavitation.

One hundred and fifty were far advanced and had class "B" symptoms. Of these thirty had positive evidence of chronic ulceration —an incidence of 20 per cent. Colonic hypermotility was present in twenty-two of these thirty cases, 73½ per cent. Sixteen of these positive cases also had positive enema evidence. Eight who had been positive with the meal examination were negative when examined by enema. Twenty had symptoms

referable to the gastro-intestinal tract and ten had none. None had gastric residue at the sixth hours. Twenty-five had pulmonary cavitation.

Twenty-six were far advanced and had class "C" symptoms. Of these twelve had positive evidence of colonic ulceration, an incidence of 45.8 per cent. Of the positive cases six had colonic hypermotility (50 per cent.) Eleven were examined by barium enema and all had positive evidence of ulceration. Ten had gastro-intestinal symptoms and two did not. Gastric residue was found after the sixth hour in two. Ten had pulmonary cavitation.

Fifty-seven classed moderately advanced and minimal cases were examined, many of whom were referred by the attending physician because of gastro-intestinal symptoms. It would appear, therefore, that the incidence of colonic ulceration, due to this partial selection of material, is somewhat higher than would be found in similar classes examined routinely.

Twenty-seven were classed moderately advanced with "A" symptoms. Three of these had evidence of colonic ulceration. All three had colonic hypermotility. Two were also positive by enema while one was not. Two had gastro-intestinal symptoms and one did not. None had gastric residue at the sixth hour. None had pulmonary cavity.

Twenty-three were classed moderately advanced with "B"symptoms. Four had evidence of colonic ulceration. Of the four positive cases three had colonic hypermotility. Three were also positive by enema while one was not. All four had symptoms referable to the gastro-intestinal tract. None had pulmonary cavity.

Twenty-three were classed moderately advanced with "B" symptoms. Four had evidence of colonic ulceration. Of the four positive cases three had colonic hypermotility. Three were also positive by enema while one was not. All four had symptoms referable to the gastro-intestinal tract. None had a gastric residue at the sixth hour. None had pulmonary cavity.

One was classed moderately advanced with "C" symptoms. This case had no evidence of colonic ulceration.

Five minimal "A" cases were examined and all were negative. One minimal "B" case was examined and found to have evidence of colonic ulceration. Colonic hypermotility was present. Gastro-intestinal symptoms were complained of. There was no gastric residue at the sixth hour. There was no pulmonary cavity.

Thirty-five had no clinical tuberculosis. None of these had evidence of colonic ulceration. Of the three hundred examined fifty-two had evidence of colonic ulceration, an incidence of 17.3 per cent. The incidence in the tuberculous examined (265) was 19.6 per cent. Thirty-seven (71 per cent) of the positive cases had colonic hypermotility. Three, or 1.2 per cent of the negative cases had colonic hypermotility. Thirty-five (67 per cent) of the fifty-two positive cases were also positive when examined by the barium enema. Ten of the cases positive by the barium meal findings were negative when examined by the barium enema. Thirty-seven of the positive cases had gastro-intestinal symptoms.

One hundred and forty-four of the negative cases also had symptoms referable to the gastro-intestinal tract.

Fifteen positive cases had no gastro-intestinal symptoms.

One hundred and four of the negative cases were also free of gastro-intestinal symptoms. Gastric residue at the sixth hour after the meal ingestion was found in three positive cases. Also in nineteen negative cases.

Pulmonary cavitation was present in thirty-seven of the positive cases (71 per cent.) One hundred and eight of the negative cases also had pulmonary cavitation.

It is of interest to note that a gastric residue at the sixth hour was found in a higher per cent of negative cases than of positive cases. This is contrary to the impression other observers have expressed. The failure of the barium enema to demonstrate colonic ulceration in many cases in which the barium meal had given positve evidence, will also be noted.

The increased incidence of positive findings amongst the more toxic groups is also shown. "A" cases 7.8 per cent, "B" cases 20 per cent, "C" cases 44.5 per cent.

The fact that 29 per cent of the positive cases did not have pulmonary cavitation should also be emphasized.

Excepting a few of the class moderately advanced, the foregoing material was non-selected and should represent an average study of a like number of similar patients in any tuberculosis group.

The chart below deals with symptoms incidence in the positive and negative studies respectively. Of the fifty-two positive cases symptoms were present in 71 per cent. Of the two hundred and forty-eight negative cases symptoms were present in 60 per cent.

	52 Positive cases	248 Negative cases
Anorexia	7.7	6.85
Nausea	7.7	10.
Gas Pains	59.6	28.6
Acid eructations	30.8	22.2
Flatulence	61.5	34.66
Undue nervousness	19.2	8-5
Unaccountable increase of constitutional symptoms	1.9	4.
Diarrhea	32.7	4.4
Constipation	11.5	29.8
Alternating diarrhea and constipation	13.5	1.2
Intestinal colic	26.9	9.7
Tenderness of abdomen	9.6	2.8
Sleeplessness	7.7	2.
Mental depression	5.8	2.
Emotional instability	15.4	4.4

An analysis of the above chart fails to indicate any clinical symptom or group of symptoms that points out conclusively the presence of intestinal ulceration.

The most common symptoms are those that result from the presence of intestinal gas. The continued presence of excessive intestinal flatulence in the tuberculous indicates the need of a study to determine the existence or non-existence of intestinal ulceration. Diarrhea, when present and continuous, is almost pathognomonic. (For purpose of this study diarrhea is defined as hypermotility, indicated by three or more bowel movements per day.) However, diarrhea will be found in only one-third of the patients having intestinal tuberculous ulcerations.

Physical signs are most often lacking. In this series abdominal tenderness was found in 9.6 per cent of the patients. As the disease progresses and approaches termination physical signs often are more frequently encountered. The abdominal muscles become spastic and very often there is a generalized abdominal tenderness.

The fact that as many as 29 per cent of positive cases in the present series were free of clinical or physical signs indicates the need of routine intestinal studies in the tuberculous.

TREATMENT

It is impossible to over emphasize the value of rest in the treatment of intestinal tuberculosis. It is this author's opinion that no form of treatment is known that will with consistency favorably influence this disease except where a regimen of rest is strictly observed. We have observed a patient who would not rest in bed except as fatigue made necessary, who had ten to twelve bowel movements daily. Following an episode of hemoptysis the patient remained in bed, was fed by a nurse and avoided all muscular activity. The bowel movements were reduced to two per day within a period of twelve days. The nearest approach to complete avoidance of muscular activity possible to institute in any active case of intestinal tuberculosis is the ideal regimen and should be continued until the signs of localized or general irritability of the colonic tract can no longer be demonstrated.

The favorable influence of general (rather than local) irradiations by the actinic rays produced by the mercury quartz vapor or carbon arc lamps are extremely gratifying. However, reliance upon this form of treatment without emphasis being placed upon the need of bed rest, is not justified. When the two are combined improvement may confidently be expected, except in the near terminal group. No attempt will be made to discuss in detail the technic of this form of treatment. The literature abounds with detailed instructions.

Abatement of symptoms and lessening of x-ray evidence of spasm and hypermotility may be expected in three to six weeks. The lessening of discomfort caused by excessive gas in the intestinal tract, which is often the only symptom present, is most often the first sign of improvement. The control of this particular symptom constitutes the greatest problem in relation to the comfort of patients.

It is often desirable to utilize ultra-violet therapy in the patient's home. Many patients find the cost of the mercury quartz vapor lamps prohibitive. In these instances the carbon arc lamp may be recommended, as the first cost is much less and treatment results compare favorably. The increased popularity of the carbon arc lamp in the last few years has been marked.

Natural heliotherapy, except in those instances where there is no pulmonary tuberculous disease, is emphatically contraindicated. Primary tuberculous infection of the intestinal tract is rarely seen, but when encountered natural heliotherapy may be considered. Use of the actinic ray should be continued for a number of months after all clinical and x-ray signs of the disease have disappeared.

The Alvarez diet which is cellulose free, easily digested, non-gas forming and of high caloric value, meets the requirements of those patients whose intestinal disease is marked by more than moderate x-ray signs of spasm and hypermotility, and having clinical manifestations, such as discomfort caused by excess gas formation, loose bowel movements, et cetera.

When clinical and x-ray signs are not so well marked a nearer approach to the usual

diet may be made, including or excluding foods with cellulose, fresh fruits, sugars and starches, as indicated.

When anorexia or poor appetite is a feature, pure olive oil may be given. It is well borne, is the easiest saponifiable of all oils, and has a high caloric value. A small amount of methyl salicylate may be added (1 c.c. to one pint of oil) to increase its palatibility. This author has prescribed alive oil both after irradiation with mercury quartz vapor lamps and without irradiation. Superiority of the irridiated product was not demonstrated.

Orange juice is usually well borne but may be alternated with tomato juice which is equally well borne and supplies qualities deficient in a strict Alvarez diet.

The nearest approach to a normal diet possible, always eliminating foods that are known to cause discomfort, is the diet of choice. Many patients resent a special diet and come to regard their food as a form of medicine and develop a psychological trend that interferes materially with appetite and digestion.

Sweet milk is often the cause of increased gas formation and resultant discomfort. Many of these patients can drink butter milk and milk in which the bacillus acidophilus has been cultured, without discomfort.

Calcium Chloride Therapy: Examination of healed tuberculous ulcers of the intestinal tract discloses that the healing process is quite different from that of tuberculous areas in the lung or glands, in that calcium deposition has no part in the healing of the intestinal ulcer. Calcium chloride administered intravenously seems to cause an amelioration of symptoms such as gas pains, intestinal colic, diarrhea, and often favorably influences the appetite. It should be remembered, however, that its value is found in its beneficent influence upon symptoms and not in its curative powers. Its greatest field of usefulness will, therefore, be found in the first few weeks of treatment, before rest and the actinic ray have themselves brought comfort to the patient. Calcium administration should rarely be indicated longer than three months. The dose is five cubic centimeters of the five per cent solution given intravenously bi-weekly. Extreme care should be taken to insure that none be deposited outside the vein because of its highly irritating effect upon tissues, which results in ulcer formation very difficult to heal.

Diarrhea that fails to subside after the above outlined treatment has been instituted or that must be checked before such treatment may become effective, is best controlled by some form of opium, preferably paregoric.

Often constipation is a prominent symptom. Laxatives should be consistently avoided because of their unfavorable influence upon the spastic and irritable areas. The use of lubricants with or without agar content is more satisfactory. Often the condition is most satisfactorily corrected by the use of olive oil or cotton seed oil (4 to 6 ounces) instilled into the rectum. Normal saline enemas may, at times, be indicated either as a method of clearing the colon or giving relief for excessive gas. In this instance, the addition of sodium bicarbonate is helpful.

The above outlined treatment will, in the majority of cases, bring about clinical and pathological improvement and sometimes cure. Almost all patients can be made comfortable. The hopeless terminal group with distressing intestinal symptoms should be given narcotics in such doses and with the frequency necessary for comfort.

Other special forms of treatment, as pneumoperitoneum or roentgen ray therapy, will rarely be needed.

Surgical interference is rarely indicated except in the hypertrophic form where occlusion of the gut lumen is threatened.

The fluid content should be carefully maintained by encouraging the patient to drink sufficient water. This is most important when the bowel movements are frequent and thin.

SUMMARY

1. Fluoroscopy for the purpose of establishing the identity of structures and authenticity of filling defects is essential.

2. Evidence obtained from the study of the twenty-fifth hour film is most important in the determination of the presence of local irritability and general colonic hypermotility.

3. X-ray evidence of local spasm in the proximal colon and generalized colonic hypermotility, when present, constitute the basis of the diagnosis of ulceration in the proximal colon.

4. Except in isolated instances a study by aid of the opaque meal will give more accurate information than that of the opaque enema.

5. Evidence obtained from a study of an opaque enema is valuable when evidence of spasm is shown. A lack of this evidence, however, does not exclude the presence of spasm.

6. A complete study of the colon must include that of the opaque enema because of

its substantiating value and the possibility of demonstration of spasm lacking in the meal result.

7. Any evidence to be of value must be demonstrable at different hours and definitely localized.

8. A definite demonstration of local spasm in the proximal colon without general colonic hypermotility is sufficient basis for positive diagnosis.

9. The incidence of intestinal tuberculosis in the far advanced class and in the toxic group of any class is such that an x-ray study of the intestinal tract is indicated.

10. The presence of symptoms not accounted for by pulmonary disease constitutes an indication for an intestinal x-ray examination of any tuberculous subject.

11. The presence of prominent intestinal symptoms are not a reliable basis for a diagnosis. In conjunction with x-ray evidence they have a certain value.

12. A positive diagnosis based upon x-ray evidence is not justified even in the absence of gastro-intestinal symptoms.

13. Best treatment results are obtained when physical rest is made the basis of the treatment plan.

14. A strict diet regime is not indicated except where the clinical and x-ray signs are rather marked.

(NOTE: The x-ray interpretative standards as applied to this series of studies were as similar to those of H. L. Sampson at Trudeau as frequent consultation and extended observation of his work could make possible. Appreciation of this help and courtesy is hereby gratefully acknowledged. It is also a pleasure to record the enthusiastic help of Dr. Harry B. Williams, Roentgenologist, at United States Veterans' Hospital, Sunmount, New York. All film interpretations and fluoroscopic observations were made in conjunction with him. He also participated in the many consultations with H. L. Sampson of Trudeau.

Appreciation is also expressed to Dr. Lawrason Brown whose criticisms and suggestions were solicited in connection with this study).

ATELECTASIS (MASSIVE COLLAPSE) IN PULMONARY TUBERCULOSIS

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(Read before the Fortieth Annual Meeting of the Arizona State Medical Association, held at Nogales, May 7-9, 1931).

The literature on atelectasis or massive collapse, occurring in pulmonary tuberculosis, is scanty indeed, even though this is a rather frequent complication during the course of the disease. The reason for this is probably that it is not generally recognized as such but is mistaken for thickened pleura, fibroid lung, tuberculous pneumonia, and at times pleural effusion. These named conditions however may be present at the same time and associated with atelectasis. Packard reported three cases occurring in tuberculous patients and states that it usually has a beneficial effect on the course of the disease. He also comments on the mildness of the symptoms and the permanency of this condition once established. Jacoboeus and Westermark, in their symposium on atelectasis, report four cases in connection with tuberculosis and show the value of artificial pneumothorax in treating these cases, as there is a tendency for uncollapsed cavities to enlarge. During the past two years at this institution we have seen a considerable number of these cases, and in nearly all of these have attempted some form of collapse therapy. In only a few was there a marked improvement in the patient's condition from the atelectasis itself. In only one was there a re-expansion of the collapsed or atelectatic lung.

PATHOLOGY

The cause of atelectasis is an obstruction or stenosis of a bronchus. When this occurs the air remaining in the lung cells, beyond the obstruction, is absorbed and, as a result, the lung tissue becomes collapsed, or atelectatic. Consequently, a marked negative pressure is produced, and, as nature abhors a vacuum, the heart, mediastinum, diaphragm, and often the opposite lung are displaced to overcome it. Where the collapse or atelectasis, remains permanent the lung becomes fibrotic in character.

A similar phenomenon occurs in artificial pneumothorax cases, where the compressed lung has become fibrotic and fails to re-expand when the pneumothorax is discontinued. Here the air in the pleural cavity is absorbed and a marked negative pressure produced, and consequently, as in the previous condition, the heart, mediastinum, opposite lung, and diaphragm are displaced to overcome this negative pressure. Fluid usually occurs in these cases, due to the decreased pressure.

What part a fibrosis of a lung in tuberculosis, with a contraction of the scar tissue, plays in producing displacement, might be a debatable question. It is possibly a factor in some of the cases where there is some aeration of the lung tissue present. However, it is more probable that most of the cases attributable to a fibrotic lung are due in reality to an atelectasis.

SYMPTOMS

We have found the symptoms produced by a massive collapse, as a rule, very mild and many times the collapse occurring without the knowledge of the patient. In the case in which the atelectasis has already been established some patients will remember a

slight hemorrhage following which they ran a mild temperature for a few days, or may state they had some pleurisy in the past. Other than this, it is hard to establish the time that the collapse occurred. In the cases that have occurred under our observation, the patients complain of some pain in the involved lung and some shortness of breath. At times the symptoms may be severe and mistaken for a pneumonia.

PHYSICAL AND ROENTGENOGRAPH FINDINGS

The physical and roentgenograph findings vary, depending on the amount of lung tissue involved and the pre-existing pathology present. The collapse, or atelectasis, may involve the entire lung, or it may be confined to one lobe or part of a lobe. Where there is considerable atelectasis, the physical findings are usually typical, namely, absence of expansion of the involved side, pulling over of the heart and mediastinum, dullness and absent breath sounds. Rales as a rule disappear, but they may become coarser and bubbling in character. At times, the breath sounds may be pneumonic in character, or amphoric if there is an underlying cavity. Cyanosis and an increase in pulse and temperature, are noted, but these usually disappear in a short time. Roentgenographically there is usually a marked opacity or consolidation, depending on the amount of lung tissue involved, with a narrowing of the intercostal spaces, drawing over of the heart, mediastinum and trachea towards the involved side, and an elevation of the diaphragm. Cavities which were present before may become obliterated or may be more distinct. Fluoroscopic examination reveals the same, but, in addition, the movement of the diaphragm is restricted.

TREATMENT

As the atelectasis tends to remain permanent, and aeration of the lung cannot be established by the usual means employed in postoperative cases, and as there is a tendency for cavities to enlarge, artificial pneumothorax seems to be the treatment of choice. In these cases the interpleural pressure is markedly reduced and the fluid is apt to be sucked over the top of the manometer if one is not careful. By instituting pneumothorax, this negative pressure is reduced, the heart and mediastinum returned to their normal position, the diaphragm depressed, and the lung still further compressed. In some of the cases where there was no free pleural space and the disease continued to progress, phrenic exeresis and thoracoplasty have been resorted to with most gratifying results. In one of our cases, a spontaneous pneumothorax occurred a short time before artificial pneumothorax was to be attempted.

CASE REPORTS

Case 1., A. O., married woman, aged 45, admitted Nov. 4, 1930. Family and past history negative. Following influenza in May, 1929, she did not regain her strength. She developed a slight cough and had intermittent pleurisy in the right chest. There was a loss of about 25 pounds in wieght. In July, 1930, she spat a mouthful of blood and later began running temperature. Sputum test positive

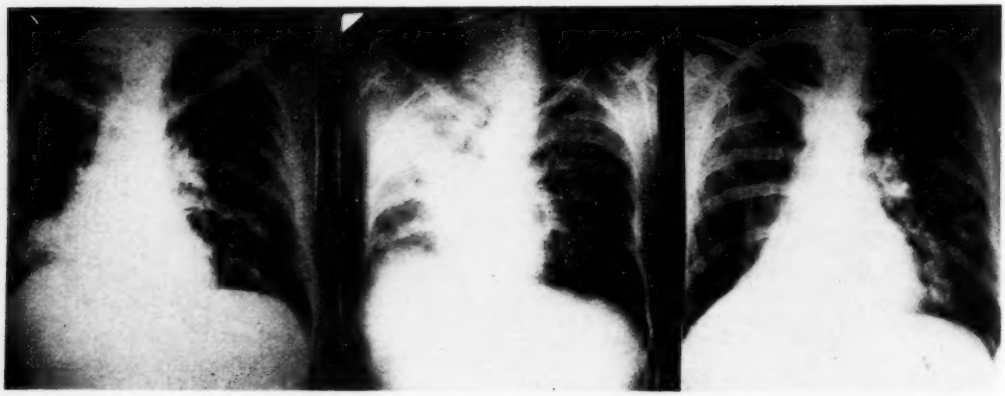

Fig. 1. Fig. 2. Fig. 3.

Fig. 1. Case I. Roentgenogram taken Jan. 12, 1931, showing slight cardiac displacement to the right, cavity in right upper lobe and opaque area in right base.

Fig. 2. Case I. Roentgenogram taken Feb. 20, 1931, two days after atelectasis developed. Shows displacement of heart and trachea, marked elevation of the diaphragm, and opacity in right chest. Cavity in upper lobe more distinct.

Fig. 3. Case I. Roentgenogram taken March 28, 1931, thirty-six days after partial pneumothorax was started. Heart, trachea and diaphragm are now in normal positions. Upper lobe held partially open by adhesions of pleura.

for tubercle bacilli. Under rest at home she did not improve.

On admission to St. Luke's Home her weight was 103. Normal weight, 145. Blood pressure: systolic, 140; diastolic, 90. Initial chest examination revealed diminished expansion of the right chest with medium rales base up. No cavity signs were made out and the percussion note was fairly resonant. Over the mid-portion of the left lung were a few scattered medium rales. An x-ray, taken Nov. 5, 1930, showed scattered infiltration, chiefly fibro-caseous, in the right upper with a suspicious, fairly large cavity. In the base and near the heart was an opaque area about 3 inches in width. The right diaphragm was slightly elevated. This opaque area was interpreted as a caseous pneumonia which might be due to a localized atelectasis. There was some fibro-caseous infiltration in the mid-portion of the left lung. Under the fluoroscope there was some restriction of the motion of the diaphragm. The Wassermann was negative and the sedimentation time was 25 minutes. Sputum positive for tubercle bacilli. Patient was placed on right postural rest. Her temperature averaged 99 to 99.4; pulse range, 90-100. X-ray taken Jan. 12, 1931 (Fig. 1), showed an increase in the pathology in the left side and the cavity was more pronounced in the right apex. The same opacity in the right base was noted as in the previous film. Physical examination did not reveal the cavity in the right apex, but there was an increase in the rales in the left lung. Patient's weight on Feb. 10, 1931, was 126, a gain of 20 pounds.

On Feb. 18, 1931, patient complained of some pain in the right lung and a slight increase in cough. The temperature was 101° and the pulse 115. There was no shortness of breath nor did she complain of palpitation. Physical examination now revealed dullness over the entire right lung except above the second rib and fourth dorsal spine. The breath sounds were now amphoric in character in the upper part of the upper lobe, below this the breath sounds and rales were absent. The left border of the heart was displaced toward the right. Patient was moderately cyanosed. X-ray taken Feb. 20, 1931 (Fig. 2), showed a massive collapse or atelectasis of the right lung confined to the lower part of the upper lobe, the entire middle and lower lobes. The heart, mediastinum, and trachea were markedly pulled over to the right, the left border of the heart being a little to the left of the spine. The right diaphragm showed considerable elevation, the dome lying in the fourth interspace. The cavity in the right apex was now exceedingly plain. The patient did not complain of much discomfort.

Artificial pneumothorax was instituted on Feb. 20, 1931. The initial manometer reading could not be obtained as the marked negative pressure drew the fluid over the top of the manometer. When 375 c.c. of air was introduced the resultant reading was minus 2 to minus 6. Under the fluoroscope, the heart and mediastinum were seen to have returned to their normal position and the diaphragm was depressed. The temperature dropped to 99° and the pulse to 80. Since then there have been a number of refills and the patient's general condition has improved somewhat. An x-ray taken March 28 1931 (Fig. 3), shows a partial pneumothorax, chiefly at the base, the upper part of the lung being held out by adhesions. The heart and diaphragm are now in their normal position. The cavity in the right upper is still present, but smaller and displaced inward. The indications now are for increasing the compression on the rgiht side.

Case II. A. B., spinster, aged 23, admitted Oct. 20, 1930. Family and past history of no importance.

Present illness began in April, 1927, with loss of weight, cough and streaked sputum. For two years she was under treatment in a sanitarium and rapidly improved. She was discharged without symptoms but still active. Since then she has not felt well and came to Phoenix in 1929. With rest she felt quite well. In May, 1930, she had a small hemorrhage. Since that time her cough has increased and she continued losing a little weight. During June, 1930, her temperature ranged from 100° to 102°.

On admission her weight was 102½ pounds. Temperature, 98°-99.2° and pulse 90-100. Chest exami-

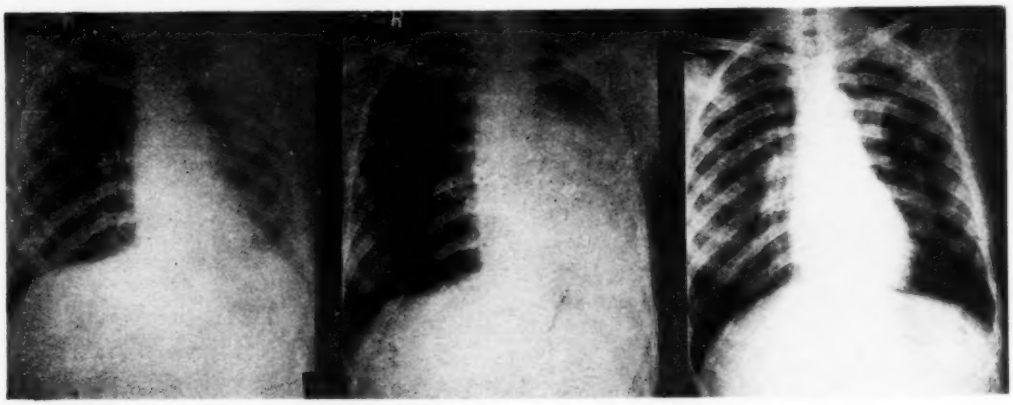

Fig. 4. Fig. 5. Fig. 6.

Fig. 4. Case II. Roentgenogram taken March 9, 1929, showing slight haziness over entire left lung, with cavity in left apex. Left diaphragm slightly elevated and heart displaced slightly to the left.

Fig. 5. Case II. Roentgenogram taken Oct. 19, 1930, showing massive atelectasis of the left lung, with open cavity in the apex. Heart displaced to the left diaphragm elevated.

Fig. 6. Case II. Roentgenogram taken Feb. 23, 1931, showing the effect of pneumothorax, with return of heart and diaphragm to their normal positions. Cavity at apex still open.

nation showed marked diminished expansion of the left chest, with dullness from the base to the second rib and fourth dorsal spine. The breath sounds were absent over the dull area but there were cavity signs in the apex. The heart was displaced to the left. Under the fluoroscope the left chest was opaque except in the apex, where there was a cavity. The left diaphragm was high and motion restricted. An x-ray taken Oct. 19, 1930, revealed the left lung opaque except for an apical cavity (Fig. 5). The heart and trachea were displaced to the left and the left diaphragm was high. A diagnosis of atelectasis of part of the upper lobe and the entire lower lobe was made. Sputum was positive for tubercle bacilli. Urine negative.

A previous x-ray, taken March 9, 1929 (Fig. 4), was obtained and this showed the heart slightly displaced to the left. The left diaphragm slightly elevated. The left lung was slightly hazy over its entirety. There was a fairly large cavity in the left apex with infiltration into the upper lobe, some being hidden by the haziness.

Pneumothorax was begun Oct. 20, 1930. The initial reading could not be obtained, due to the marked negative pressure drawing the fluid over the top of the manometer. When 300 c.c. of air was given the resultant reading was minus 3 to minus 5. Under the fluoroscope the heart and diaphragm returned to their normal position and the lung considerably compressed. Since then the patient has been taking pneumothorax regularly and has shown considerable improvement, even though the cavity in the left apex is still present. She was discharged Nov. 17, 1930, to her home. Her temperature was normal and pulse reduced. Her weight was 109.

X-ray taken Feb. 23, 1931 (Fig. 6) shows heart and trachea and diaphragm in normal position. The left lung is about collapsed but the cavity in the apex is still held open by adhesions. Thoracoplasty will probably be necessary for a closure of the cavity.

This case is interesting in that no definite history could be obtained as to the time of the massive collapse. It probably occurred at the time she had her hemorrhage. Also because the x-ray taken in March, 1929, shows a partial atelectasis in that the heart was slightly displaced to the left and the diaphragm slightly elevated; also, the left lung was hazy.

Case III. C. S., minister, aged 72, admitted Nov. 6, 1930. Family history negative. At 30 years of age he had a "breakdown." Rested for a while and later took up engineering work, which he continued for 15 years. Felt fairly well most of the time. In 1912, after he had returned to the ministry, began losing weight and coughing. No tuberculosis found at this time and the patient rested at home. His general condition was fair but he would have intermittent hemorrhage.

On admission patient was fairly well nourished, weight was 150. Temperature range, 99°-100°; pulse, 95-110. Blood pressure: systolic, 145; diastolic, 95. Chest examination showed cavity signs in the right apex and scattered medium rales over the right upper chest. There were a few scattered rales in the left apex. Patient was instructed to take right postural rest. X-ray taken Nov. 8, 1930 (Fig. 7), showed marked fibrosis in the right apex with a cavity. There was some scattered infiltration in the rest of the right lung. Left lung showed some fibrosis. The heart was slightly displaced to the right. Sputum positive for tubercle bacilli.

On Nov. 23, 1930, patient began hemorrhaging. Usual methods did not control it. Pneumothorax was attempted but there was no free pleural space. On Nov. 25, 1930, a blood transfusion was done and a phrenic exeresis, but small hemorrhages continued. Dec. 3, 1930, patient developed pain in the right chest, dyspnea and some cyanosis. Chest examination showed absent expansion on the right side, with dullness. The breath sounds were absent, and the heart displaced to the right. X-ray taken Dec. 4, 1930 (Fig. 8) showed a massive collapse of the right lung with marked cardiac displacement. The diaphragm was high, probably due to both the phrenicetomy and the atelectasis. Patient was quite ill and at times delirious. Had considerable mediastinal pain. On Dec. 18, 1930, the breath sounds were coming through on the right side but the heart was still displaced. Dullness was still present. Daily examination showed the

Fig. 7. Fig. 8. Fig. 9.

Fig. 7. Case III. Roentgenogram taken Nov. 8, 1930, showing marked fibrosis with cavity at right apex.

Fig. 8. Case III. Roentgenogram taken Dec. 4, 1930, showing massive atelectasis of the right lung, with marked displacement of the heart.

Fig. 9. Case III. Roentgenogram taken Dec. 26, 1930, showing re-expansion of the right lung and heart returned nearly to its normal position. There are areas of caseous bronchopneumonia in the right base. High diaphragm is due to phrenic exeresis on the right.

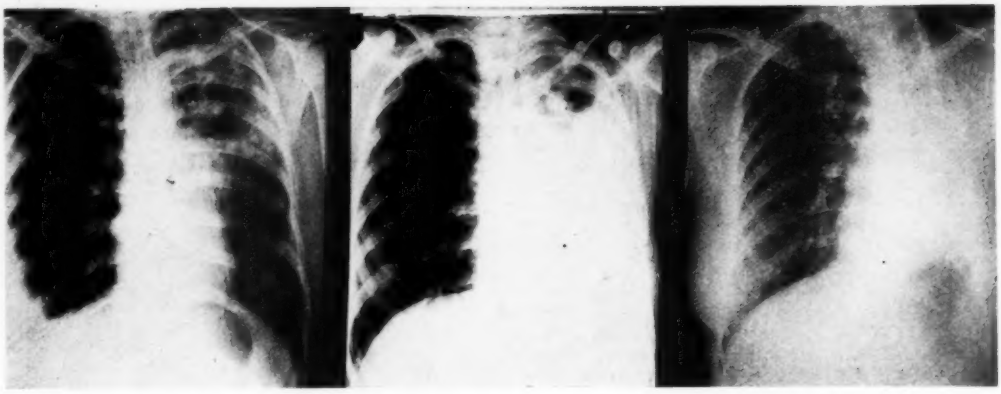

Fig. 10. Fig. 11. Fig. 12.

Fig. 10. Case IV. Roentgenogram taken July 5, 1929, showing infiltration in the left upper with cavity.

Fig. 11. Case IV. Roentgenogram taken Aug. 26, 1929, showing massive atelectasis of the left lung. Large cavity in apex. Heart displaced to the left. Diaphragm high, as seen under fluorosco pe.

Fig. 12. Case IV. Roentgenogram taken Feb. 5, 1931, showing conditions after a posterior extrapleural thoracoplasty.

right lung clearing and an x-ray taken Dec. 28, 1930 (Fig. 9) showed the atelectasis cleared and the heart returned to its normal position. The right diaphragm was high, due to the phrenic intervention. There was some caseous broncho-pneumonia remaining in the lower right.

At the time of the atelectasis the temperature rose to 102.5° and continued for about four days, when it fell to about 99°, where it remained. The pulse rose to 120 and gradually returned to 90-100. Later the pulse ranged between 110 and 120. The patient's condition steadily grew worse in spite of the clearing of the collapse. He had considerable cardiac pain. The right lung did not show any further change by physical examination. Patient died Jan. 3, 1931. Autopsy was not obtained.

Case IV. D. B., housewife, aged 27, admitted Aug. 15, 1929. Grandmother died of tuberculosis. Past history. She had the usual childhood diseases. Always well and strong. Appendectomy, 1924. Ether anesthesis. Complete recovery. She has one child, age 6, living and well.

In February 1929, following the influenza, she did not regain her strength. Coughed considerably. In June she began having night sweats and cough increased. Consulted a physician in July, 1929, and was told that she had tuberculosis. An x-ray taken July 5, 1929 (Fig. 10), showed considerable scattered caseation above the fourth rib, with a cavity in the apex. There was no history of pain, any marked rise in temperature, not shortness of breath.

On admission, chest examination revealed dullness, base up on the left with diminished or absent breath sounds. There were cavity signs present in the left apex, with a few large rales in the upper chest. Fluoroscopic examination showed a marked opacity over the left lung with the exception of the apex, where there was a large cavity. The heart was pulled to the left and the diaphragm was high and fixed. A diagnosis of atelectasis was made. X-ray taken Aug. 26, 1929 (Fig. 11), revealed the same as the fluoroscope, only that the diaphragm was not well made out. Sputum examination was positive for tubercle bacilli. The temperature range was 99°-101° and the pulse 80-100. She was placed on left postural rest, and later pneumothorax was attempted but there was no free pleural space nor was any fluid obtained at the time. Under rest her temperature and pulse subsided somewhat and she gained weight. A phrenic exeresis was done on the left side Jan. 14, 1930, and an x-ray in March of the same year showed the cavity in the left apex greatly enlarging, the lung still remaining collapsed. In May, 1930, a left-sided thoracoplasty was done and since that time, the patient has made remarkable improvement. At present she is free from cough and temperature and her weight is 130. Sputum still positive. X-ray taken Feb. 5, 1931, (Fig. 12) shows a complete thoracoplasty on the left side. The right lung remaining clear.

The atelectasis in this case occurred between July 5, 1929, and her admission on August 26, 1929. Further questioning of patient did not reveal exact time of the atelectasis.

Case V., E. N., housewife, aged 32, admitted Oct. 14, 1930. Family and past history negative.

In 1928 she began coughing and running temperature. She also had some pain in both chests. In December, 1929, a positive sputum was found and she was told that she had tuberculosis. Since that time she has been under treatment in a number of sanitariums and has improved somewhat. For the past few months she has had periodic upsets with high temperature which lasts for a few days. Sputum decreased at these times.

On admission was normal except for an occasional rise. The pulse range was 80-90. Sputum positive for tubercle bacilli. Blood Wassermann negative and the sedimentation time 3 hours. Her weight was 128. Normal weight, 130. Chest examination revealed dullness, base up on the left side, with diminished breath sounds. There were crackles over the left chest. There were a few scattered rales over the right upper. Fluoroscopic examination showed the heart displaced to the left, the left diaphragm high and motion restricted. The left chest was more or less opaque, with areas of lesser density. Roentgenogram dated Oct. 16, 1930, showed a marked density throughout the left lung, in which could be seen a fairly large cavity. The heart was displaced to the left and the diaphragm was high. Findings typical of atelectasis.

Artificial pneumothorax was attempted a number of times in this case but was not successful. Fluid or pus was also looked for at these times. Patient continued having periodical upsets with high temperature, 102°-104°, at intervals and thoracoplasty was recommended but refused. A phrenic exeresis was done Nov. 12, 1930, on the left side. There was no material rise in the diaphragm. Since that time her condition has remained about the same except for these periodical upsets which are probably a question of drainage of a cavity. Thoracoplasty seems to be the treatment of choice in this case.

No history was obtainable in this case as to the time that the atelectasis occurred and previous x-rays were not obtainable.

Case VI. E. K., auto-mechanic, aged 42. Family history: Three brothers died of pulmonary tuberculosis. Past history: Herniotomy, bilateral, 1914, ether anesthesia. States that he has been coughing for 20 years. Present illness began in 1926 with cough, temperature and loss of weight. A positive sputum was found at this time and since then he has been resting and feeling better. For the past year he has had gastric upsets with vomiting after eating. His cough has been moderately severe and he expectorates about 2 ounces a day. For the past few months his stomach has given him considerable trouble and he came for relief of this.

Physical examination, March 27th, 1931, showed diminished expansion over the left chest. In the base of the left lung there was dullness. The breath sounds were tubular in character over the entire left chest. Whispered pectoriloquy was present over the left lung. There were scattered medium coarse rales over the base. In the right apex were a few medium rales. Fluoroscopic examination showed the heart displaced to the left, the diaphragm high and restricted in motion. In the base of the left lung was a marked opacity, above this there was a large cavity or multiple cavities. Abdominal examination did not reveal any pain or tumor masses. An x-ray (Fig. 13) taken the same day revealed the

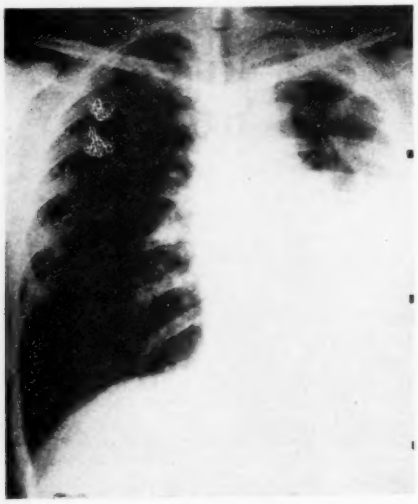

Fig. 13.

Fig 13. Case VI. Roentgenogram taken March 27, 1930, showing atelectasis in the left base, with large cavities in the left upper. Under fluoroscope diaphragm seen to be very high.

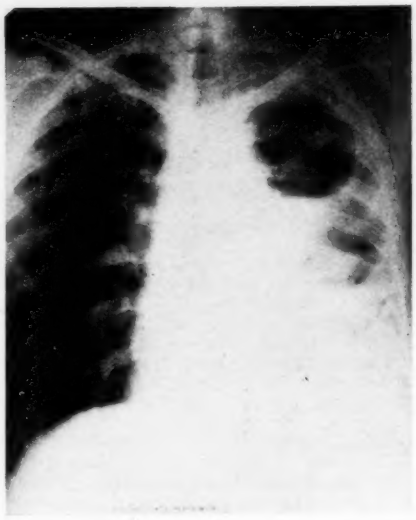

Fig. 14.

Fig. 14. Case VI. Roentgenogram taken April 5, 1930, showing spontaneous pneumothorax which had developed. The heart has returned to its normal position.

same as the fluoroscope. A diagnosis of atelectasis in the left base with a large cavity in the upper part of the left lung, was made. Thoracoplasty was advised in this case providing a gastro-intestinal examination was negative. Patient wished to try pneumothorax first. He was to enter the hospital on April 14, 1930, for an attempt at artificial pneumothorax but the night before he developed a sudden pain over the left chest with shortness of breath. A diagnosis of spontaneous pneumothorax was made. An x-ray taken the following day, April 5, 1930 (Fig. 14) showed the heart displaced slightly to the right and an area of pneumothorax in the left lower. Aspiration had to be done later because of the fluid and dyspnea and this showed a beginning empyema. Later a rib resection was resorted to and this was later followed by a phrenic exeresis and a thoracoplasty. Patient died later from shock of an anterior rib resection.

Case VII. H. E., housewife, aged 32. Mother and two brothers died of tuberculosis. Past illness of no importance.

In 1918 patient had influenza. Following this she continued coughing and had a slight hemorrhage. For a time took the rest cure and improved. Did her own housework until 1928, when she began running temperature, coughing and losing weight. Since that time she has been confined to bed but has not noticed any improvement in her condition. She has had pains in the left chest. Chest examination made on July 3, 1929, showed a few medium rales in the right upper chest. In the left base there was considerable impairment in the resonance and diminished breath sounds. In the left apex there was increased whispered voice. Coarse rales were present over the entire lung field. The heart was displaced to the left. Her temperature ranged from 80° to 100° and she expectorated about 4 ounces daily.

She had previously had an x-ray made, Feb. 6, 1928, which showed some scattered infiltration in the right upper lung. The heart and trachea were slightly displaced to the left, and the left diaphragm

possibly showed a slight elevation. The left lung was slightly hazy throughout and showed scattered infiltration and a large cavity in the mid-portion. X-ray taken July 1, 1929, showed an atelectasis in the left base with heart displaced to the left and the diaphragm high. There were large cavities present in the left upper and a small cavity in the mid-portion of the right lung.

Artificial pneumothorax was started on the left side on July 20, 1929. The initial reading was minus 9 minus 4. Two hundred cubic centimeters of air was given and the resultant readings were minus 5 minus 3. After a number of refills, fluoroscopic examination showed a partial collapse of the left lung, the heart and diaphragm in their normal position. An x-ray taken Jan. 2, 1930, showed a partial compression of the left lung with two open cavities; heart and diaphragm in normal position. The right lung showed the cavity in the mid-portion greatly increased in size. Pneumothorax was continued, however, as the patient was more comfortable and as re-expansion of the left lung would not take place.

Case VIII. H. M., bachelor, aged 35, admitted Dec. 8, 1929. Family history negative.

Appendectomy, April 1923, and never felt well afterwards. Ether anesthesia. In February, 1926, began losing weight and having night sweats. Sputum examination at this time was positive and he was placed on bed rest. Has been under treatment continually and never placed on exercise. His weight continued to fall off and he continued running temperature.

Physical examination, Dec. 8, 1929, showed him to be markedly undernourished. Weight was 121; normal weight, 180. His temperature range, 98°-99.6°. Pulse 84 to 90. He was raising 4 ounces of sputum a day, which was positive for tubercle bacilli. Chest examination showed medium rales above the fourth rib and fourth dorsal spine on the right. On the left there were medium coarse rales, base up, and increased whispered voice in the apex.

Roentgenograph taken Dec. 11, 1929 (Fig. 15) showed fibro-caseous infiltration in the right upper above the second rib, with a questionable cavity. The left showed multiple cavities over the entire lung.

Under rest there was slight improvement, so a phrenic exeresis was done April 24, 1920. There was a good rise in the diaphragm. In June, 1930, patient went to Los Angeles, where he continued under treatment. X-ray, July 18, 1930, (Fig. 16) showed cavities still open on the left and the left diaphragm high. There was a slow gain in weight, In October 1930, his weight was 140. On Oct. 15, 1930, patient had a moderated hemorrhage. Following this, temperature was elevated and he had some soreness in the left chest. He soon began to feel better and gained weight rapidly. In January 1931, patient returned to Phoenix. Weight at that time was 155. Chest examination at that time showed dullness over entire left chest with markedly increased whispered voice and bronchial type of breathing. There were a few scattered rales over the left chest and in the right apex. An x-ray taken Jan. 20, 1931 (Fig. 17) showed the pathology in the right apex decreased considerably. On the left there was a total opacity with an extremely elevated diaphragm. The heart was displaced to the left. This opacity over the left was undoubtedly an atelectasis which occurred at the time of his hemorrhage in October 1930. Since that time he has shown a very marked improvement, with normal temperature and pulse, and his present weight is 178, a gain of 38 pounds following the collapse. Intervention was not deemed necessary in this case thus far.

SUMMARY

Atelectasis is more common in pulmonary tuberculosis than is generally recognized and, once established, it is usually permanent. The symptoms may be mild and often overlooked by the patient. In some cases the tuberculosis shows a marked improvement from the atelectasis, but it is probably more apt to be detrimental, because of the cardiac displacement and the tendency for cavities to enlarge. The treatment of choice is artificial pneumothorax. Atelectasis should be considered in all cases where the roentgenogram shows a narrowing of

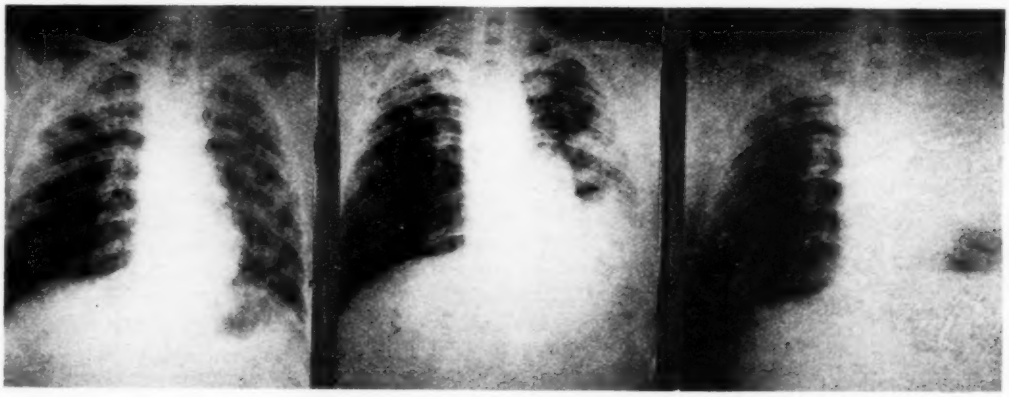

Fig. 15. Fig. 16. Fig. 17.

Fig. 15. Case VIII. Roentgenogram taken Dec. 11, 1929, showing infiltration through right upper and large cavities throughout left lung field.

Fig. 16. Case VIII. Roentgenogram taken July 18, 1930, showing effect of phrenic exeresis on the left side.

Fig. 17. Case VIII. Roentgenogram taken Jan. 20, 1931, showing atelectasis of the entire left lung.

one side with cardiac displacement and an elevation of the diaphragm, regardless of the amount of opacity or density in the affected side.

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DISCUSSION

DR. HOWELL RANDOLPH, (Phoenix): The surgical literature of the past five years has been filled with articles calling attention to massive atelectasis. Among postoperative complications it has come to outrank pneumonia in the minds of many. Coryllos goes so far as to state that between 50 and 75 per cent of so-called postoperative pneumonias are at some stage purely cases of atelectasis. This paper is one of the few calling attention to the occurrence of the condition in exudative or suppurative pulmonary disease, diseases in which the conditions now thought responsible for the production of atelectasis are so often present, namely, bronchial obstruction by infectious material, together with coughing and difficult breathing, at times the bronchial plug acting as a valve. When considering this mechanism of production it seems most strange that more typical cases of massive collapse are not seen in pulmonary tuberculosis. I am highly indebted to Dr. Gatterdam for directing our attention to this subject. There is no doubt in my mind that we will see more cases if we look for them.

DR. E. A. GATTERDAM, (closing):-

In regard to the bronchoscope for relief of an atelectatsis: I have had no experience. Most of the cases seen in tuberculosis are usually of long standing so that fibrosis of the lung has occurred. Atelectasis is probably one of nature's defenses to aid the healing of a tuberculous lesion. I believe the safest and best method is to further compress the lung with pneumothorax, when possible.

The term "thickened pleura" in an x-ray reading should be used much less frequently. Roentgenologically, thickened pleura is, as a rule, hard to demrisies, such as adhesions to the diaphragm and obliteration of the costophrenic angles. It takes an extremely thickened pleura to cause a marked opacity, and many of these are due to an encysted empyema or a caseous pleurisy. Fluid is easily ruled out in these cases, as with fluid there is apt to be displacement of the heart and mediastinum to the opposite side and the diaphragm is depressed. Aspiration should be done if there is doubt.

A CRITICISM UPON THE CURRENT CONCEPTION OF ARREST IN PULMONARY TUBERCULOSIS

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A questionnaire sent to an average group of phthisiologists of the United States shows that there is a great difference of opinion as to what constitutes an arrest in pulmonary tuberculosis. Of those who replied, 40 per cent stated that they unqualifiedly followed the classification of the American Sanatorium Association, 44 per cent followed it with exceptions and reservations, while 16 per cent based their determination of arrest on other criteria.

The American Sanatorium Association first adopted a classification in 1909. Modifications have been made several times, the last being in 1922, when the roentgen ray was admitted into the classification. Since 1922, there have been many advances in our knowledge of pulmonary tuberculosis. We have, therefore, been trying to use a classification several years behind the times—a classification which does not at all coincide with the more recent developments.

The advancement of our knowledge of pulmonary tuberculosis, which must be taken into consideration in a future scheme of classification, will be briefly stated as follows:

1. Our changed viewpoint in regard to rales:

Bruns,[1] after several years of research, has come to the conclusion that the most unusual type of rales heard in pulmonary tuberculosis—those heard on inspiration, occurring in showers following an expiratory cough—are not due to moisture in the terminal bronchioles, as usually supposed. After careful experimentation on the cadaver, Bruns believes that the rales in question are due to partially collapsed alveoli or lobules, the collapse being caused by infiltrating tubercles in a neighboring lobule, by contraction of fibrous bands, by pleural adhesions, and so forth. He further found the same type of rales present in all the different anatomical-pathological types of tuberculosis; also, in cases with fever or without fever, with expectoration or without expectoration, and in arrested cases, as well as active cases. Bruns concludes, therefore, that these rales, which he calls parenchymal rales, do not necessarily mean active tuberculosis or even tuberculosis at all, are not due to moisture, and are more of the nature of atelectatic rales.

Heise[2], of Saranac, has recently published some very timely articles on rales. In one of these articles, it was his object to show that the increase or diminution of rales in pulmonary tuberculosis was of no value in determining the patient's progress. His conclusions were drawn from the study of 412 records over a six months' period. He divided his cases into four classes:

Class A: When rales over a period of six months remained the same as to area and character—111 cases

103, or 93 per cent, showed x-ray improvement;
2, or 2 per cent, showed x-ray increase;
6, or 5 per cent, showed no change.

Class B: When rales were found over a greater area—92 cases

65, or 71 per cent, showed x-ray improvement;
5, or 6 per cent, showed no change;
22, or 23 per cent, showed x-ray increase.

In Class B, if the clinician had come to the conclusion that the patients were worse be-

cause they had an increase in rales, he would have been 23 per cent right and 77 per cent wrong.

Class C: When rales showed a diminution of area—83 cases

73, or 88 per cent, showed x-ray improvement;
5, or 6 per cent, showed x-ray increase;
5, or 6 per cent, showed no change.

Class D: When rales appeared after treatment and not before—51 cases

40, or 78 per cent, showed x-ray improvement;
3, or 6 per cent, showed x-ray increase;
8, or 16 per cent, showed no change.

In Class D, a clinician going on the theory that the appearance of rales means progression of disease would have been 94 per cent wrong and 6 per cent right.

Our conclusion must, therefore, be that the increase or diminution in rales in pulmonary tuberculosis is of very limited value in depicting the anatomical changes which take p'ace in the pulmonary lesions.

2. Our changed viewpoint in regard to the value of constitutional symptoms in determining activity of pulmonary tuberculosis:

Much has been written during the last few years in regard to latent pulmonary tuberculosis in children. Such children look well, feel well, have not lost weight, have no elevation of temperature, have no physical signs of disease, but roentgenograms show marked pulmonary infiltration. McPhedran[4] remarks that, although the lesions are latent, they are usually active and often rapidly progressive—elevation of temperature, malaise, and so forth being manifestations of the disease.

What is true of children as to latency is also true of beginning tuberculosis in adults, although to a somewhat less degree. Both the acinous nodose and broncho-pneumonic types of the disease often develop very slowly. No constitutional symptoms are present or they are so slight as to escape notice, a parenchymal infiltration seen by the roentgen ray being the only evidence that the individual is ill. Here also, as in children, if proper treatment is not instituted, the lesion usually progresses and late manifestations develop.

Again, when an active case of pulmonary tuberculosis, with loss of weight, elevation of temperature, rapid pulse, and so forth, is placed on continuous rest in bed, usually in a few months a great change occurs. There is gain in weight, the temperature and pulse become normal, and practically all symptoms disappear. This startling change has been our pitfall in the past. We formerly believed, and the idea still persists in some quarters, that pulmonary tuberculosis may be arrested in a few months. The roentgen ray has demonstrated that, although clinical symptoms may disappear in a few months, the tuberculous lung pathology clears and fibroses very slowly and heals only after years of prolonged and continuous rest. It is, therefore, not only recognized that a lesion which is progressive must be considered active but also that a lesion which is retrogressing must be considered active, whether there are constitutional symptoms or not.

3. Our present ideas as to the value of the roentgen ray in diagnosis and in determining progress:

During the early part of the present decade, the roentgen ray had not even been admitted into the schema of classification of the American Sanatorium Association. Physical findings, especially rales, were considered of prime importance. Amberson's[5] articles published in 1921 and 1922 were most timely. He stressed the importance of the roentgen ray not only in diagnosis but advocated the use of stereo-roentgenograms taken serially at two to four months' interva's in order to determine the progression or retrogression of the lesions in the lungs. He made plain the point that activity could be demonstrated, not by viewing one x-ray plate alone, but by determining if changes in the lesion were taking place by the comparison of a series of roentgenograms. The medical profession has been somewhat slow in accepting Amberson's views, but at present the leading clinicians and phthisiologists place their main reliance on the roentgen ray, not only in diagnosis but also in determining the exact changes occurring in the lungs during treatment.

Gerald B. Webb[6] states that the development of the x-ray in diagnosis and its use for the exact following of changes during treatment in tuberculosis is one of the most important developments of knowledge during the last quarter of a century.

McPhedran[3] states that there is no method that records the tuberculous invasion of the lungs and the progress or retrogression with accuracy approaching that of the x-ray. He states further: "The x-ray is the method of precision for detecting the presence and evaluating the exact severity and direction of changes of a lesion. The roentgenogram is the pivotal point of early diagnosis and the guide and index of effective treatment."

4. Our increased appreciation of the value of rest in pulmonary tuberculosis:

Of all the alleged specifics and forms of treatment that have been advocated, rest a'one has endured. At the beginning of the decade, patients were required to rest only when they had an elevation of temperature. After their temperature had reached normal limits for a few weeks, toxemia had disappeared, and they had begun to gain weight, they were placed on graduated exercise. As stated above, clinicians, after adopting the study of serial roentgenograms, found how slow the clearing up of lung pathology was in comparison with the improvement of the general condition of the patient. It seemed, therefore, only logical to continue rest until an anatomical arrest had been attained, not merely a symptomatic arrest.

Still further study of roentgenograms has shown what wonderful results can be obtained by continuance of prolonged rest without interruption, especially when cavitation exists.

Thus, rest at the present time is considered more than ever a specific for the cure of pulmonary tuberculosis.

Graduated exercise, on the other hand, has come into disrepute except when the pulmonary lesion has healed; only then should a patient be prescribed exercise.

5. Certain laboratory procedures developed in the last few years are considered by many a distinct advance in determining the physical condition of the tuberculous patient:

Among the most important of these are the blood sedimentation test as developed by Fahraeus[5], Greisheimer[7], and Cutler[8], and the new significance placed on the leucocyte count in pulmonary tuberculosis, as described by Medlar[8], Flinn[10], and others.

With the above advances in our knowledge in mind, what are the criteria for a working classification in those essentials which will not only help clinicians in determining an arrest but will be a standard for all to follow?

Such criteria must be easily understood. The wording must not be ambiguous and contradictory, as found in our present classification, but should be concise and to the point.

The following suggestions are based on the present developments of our knowledge of tuberculosis, aided by answers to a questionnaire which was sent to many of the foremost phthisiologists of the country.

In this discussion, an arrested case and a healed lesion are considered synonymous. In the past, there has been too much guess work as to what constitutes an arrested case. Some clinicians, as noted in our questionnaire, consider a tuberculous patient arrested when he seems clinically well and has no bacilli in his sputum, whether this condition has lasted for a month or a year. Others would consider a case arrested only when a well-fibrosed stationary lesion, as seen by serial roentgenograms, has existed for two years. The first group is evidently confusing arrest with latency. A latent case, as stated above, may be active.

Our endeavor in the treatment of pulmonary tuberculosis is to cure our patients, not merely to alleviate symptoms and send them back to work only to have them return in worse condition than before. Sanatoriums which send out patients with a symptomatic arrest only, report that from 25 per cent to 50 per cent of such patients are dead within five years. We must, therefore, have in mind an anatomical arrest, not a symptomatic arrest. This view should be the basis of a classification.

1. Constitutional Symptoms:

In the past, constitutional symptoms have been considered very important in determining activity. This is a very crude method, and it is equally crude to state that the absence of constitutional symptoms indicates quiescence or the absence of activity. Elevation of temperature, rapid pulse, and so forth, may be due to other causes and the active case under treatment may have a normal pulse and temperature. The schema of classification of the American Sanitorium Association states as follows: "Arrested — All constitutional symptoms and expectoration with bacilli absent for a period of six months." The author of the above statement must have had in mind a symptomatic arrest, as it would be quite unusual to have an anatomical arrest within six months after all constitutional symptoms had disappeared. Also, if the criteria for arrest, as determined by the roentgen ray as suggested in the next few pages, are followed, it would be quite unusual to have constitutional symptoms enter the picture, as they are usually absent for years before an arrest is reached.

We would suggest the following as to constitutional symptoms: All constitutional symptoms that are reasonably certain to be due to tuberculosis and bacilli in sputum to be absent for at least one year. That constitutional symptoms are absent does not necessarily mean quiescence or inactivity. Such cases must be checked up by serial roentgenograms, which should be the deciding factor.

In the present scheme of classification, a

quiescent case is described as follows: "Absence of all constitutional symptoms, expectoration and bacilli may or may not be absent; physical signs and roentgen findings to be those of a stationary or retrogressive lesion, the foregoing to have existed for at least two months." Under the above definition, it is entirely possible and probable for a quiescent case, as above described, to have extensive parenchymal infiltration with cavitation. The term quiescent, therefore, as described by the present classification, only means there are no constitutional symptoms. Such cases are usually anatomically active and require years of treatment. It is illogical to set a time limit and expect an arrest in six months. Why, therefore, call them quiescent? We doubt if this term should be used in a future classification.

2. Physical Findings:

If we are to accept the newer teachings of Bruns and Heise as to physcial findings, and especially as to rales, we can come to but one conclusion. Physical findings have no place in a schema of classification as a criterion for the determination of an arrest in pulmonary tuberculosis.

3. The Roentgen Ray:

The present schema of classification gives the following x-ray definition of a healed lesion: "Roentgenograms to show clearing, fibrosis, calcification, or a stationary condition."

This statement is very ambiguous and has caused much controversy and misunderstanding, as is very evident from the answers to the questionnaire. Does it mean that clearing is still going on or is it completed? It is well to note the "or." Does it mean that we have two alternatives on which we may determine an arrest—clearing fibrosis and calcification on one hand; a stationary condition on the other? These are very vital points, and are not made clear and it is impossible to guess the intention of the authors.

*While clearing of the tuberculous lesion continues, as seen by comparison of roentgenograms, we must assume that the lesion is still unstable and activity exists. The majority of the clinicians who answered the questionnaire are of that opinion.

McPhedran[1] expresses this idea as follows: "A patient in whom pulmonary infiltration appears as soft ill-delimited spots, especially if these tend to confluence, should be treated by rest in bed until the futility of such care is proved by adequate observation. So long as such a lesion, roentgenographically recorded at three months' intervals, continues to show a material retrogression from one quarterly record to the next, the patient must be regarded as one who is still in need of the benefits of rest in bed. Such a lesion is clearly very unstable."

On the other hand, can we assume when retrogression has come to an apparent standstill, as seen by comparison of the roentgenograms, that healing has taken place? Other factors must enter the picture. The lesion must not only have become stable but it must appear hard, in other words, fibrosed.

Another factor is time. We are not justified in saying that a lesion, even if hard, is healed the first time a quarterly interval comparison of roentgenograms shows a stationary condition. The stable condition should continue for some months. McPhedran[1] supports this view. He states: "The best evidence of a healed lesion is to be found in the sharp dense strands which remain stable for months under competent roentgenographic control."

With our present knowledge of tuberculosis and backed by the authorities quoted, it is only logical to include in the roentgen ray criteria for a healed lesion the following:

*(1) A stable or stationary lesion.

(2) A hard, well-fibrosed lesion.

(3) A lesion which has remained stationary or stable for months.

(4) The above three points to have been determined by a competent roentgenologist using a proper technic and a satisfactory roentgen ray plant.

Some of the clinicians of our questionnaire say that a time requirement should not be arbitrary. Others believe a time requirement advisable. The time as suggested varied from six months to three years, the average time being thirteen months. Forty per cent are of the opinion that six months is

*Clearing must be distinguished from contraction of fibrosis. Clearing occurs as retrogression progresses and the appearance of the lesion is more or less soft. When the lesion becomes delimited and hard, changes may take place slowly, extending over some years, but these changes are usually due to contraction of fibrosis.

*There is one type of healing not included in the four points mentioned above. That is the type which is entirely absorbed, leaving no trace, and is usually of the exudative type. This occurs very frequently. We believe such cases should go through a twelve-months' period of observation and exercise before considering them arrested.

sufficient. Twenty per cent suggested that one year be the time, while twenty-five per cent believe the time should be two or more years. No statistics are available to throw light on this point and only the study of a long series of cases, intelligently followed up, can give us the answer. When in doubt, it would seem more reasonable not to adopt too short a time, as a reactivation is a serious mishap and may not only take years to rectify but may also mean the death warrant of the patient. We believe a schema of classification would not go far wrong if one year were adopted, the last six months to be the try-out period.

4. A Plan for Graduated Exercise to be included in the Schema of Classification:

Our present schema of classification offers nothing in the way of a try-out to determine if arrest is complete before patients are discharged to go back to their former life. The questionnaire shows that a few place their main reliance in determining arrest on what they call the physical capacity of the patient and practically ignore the roentgen ray findings. They begin graduated exercise early, long before a healed lesion has occurred. I have seen many cases develop extensive extensions from such practice and am inclined to agree with Landis[12] who states that "when we have become convinced that the pulmonary process has become healed, then and not until then may the patient be given exercise."

The try-out period, however, with graduated exercise, is so important that we believe it should comprise a part of a future schema of classification.

We would recommend that when a tuberculous lesion, by comparison of serial roentgenograms, appears fibrosed and remains stable for six months, the patient be required to take graduated exercise for three months and, finally, the last three months be spent as nearly as possible under the patient's probable future conditions of living.

5. The Blood Sedimentation Test and the Leucocyte Count as Criteria for the Determination of Arrest:

Felix Baum[13] believes that the blood sedimentation test is above everything else in value for determination of a healed lesion.

McPhedran[14] believes that the sedimentation test and monocyte-neutrophile lymphocyte count may emphasize retrogression but if absent should not nullify roentgen ray evidence of instability.

Although the above laboratory procedures are no doubt of considerable value in determining stability and instability in pulmonary tuberculosis, we are hardly ready to include them in a schema of classification.

Another deficiency of the classification of the American Sanatorium Association is that it is based almost entirely on the extent of the pulmonary involvement. We believe there should be combined with it an anatomico-pathological classification. Since the increase of the importance of the roentgen ray, we are becoming more and more able to differentiate the well-known types of lung pathology. A quantitative classification is no longer sufficient. It is very essential that we know the type of tuberculosis present, not only for prognosis, treatment, and statistics, but for affording a common nomenclature for the clinician, roentgenologist, and pathologist.

SUMMARY

1. The present classification of the American Sanatorium Association has never been satisfactory on account of its ambiguousness, and owing to the advances made in our knowledge of pulmonary tuberculosis during the last ten years, it is doubly unsatisfactory.

2. On account of the latency of pulmonary tuberculosis and the well-being of patients following rest, without corresponding changes in the anatomical condition of the lungs, lack of constitutional symptoms should receive but slight consideration in determining an arrest. On the other hand, constitutional symptoms, if found to be due to tuberculosis, may be considered an expression of activity.

3. As it seems to have been conclusively demonstrated that physical findings, especially rales, are of no value in determining progression and retrogression of a tuberculous lesion, it follows that they have no place in a scheme of classification.

4. The roentgen ray must be considered the main reliance in determining an arrest or a healed lesion and should receive this place of prominence in a schema of classification. When by the comparison of serial roentgenograms at quarterly intervals, a lesion is found hard, fibrosed, and has remained stable for six months, we consider that an arrest has been attained provided an additional six months is passed with carefully graduated exercise and observation without reactivation of the disease.

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ZINC CHLORIDE IN CANCER.—The use of zinc chloride and similar caustics in the treatment of cancer has been practically abandoned by all except the "cancer cure" quacks. Such caustics are difficult of control, so that there is destruction of healthy, as well as of malignant, tissue; their action is slow, thus unnecessarily prolonging the pain of removal. Much better results can be obtained by the judicious use of surgery when the growth is operable, or by the use of radium and roentgen rays when the disease has advanced beyond the chance of its complete removal. The use of such caustics has been abandoned also by the progressive dermatologists in the treatment of small epitheliomas of low malignancy; they use, instead, radium, roentgen rays, or coagulation with high frequency currents. (Jour. A. M. A., March 7, 1931, p. 797.)

THYMOPHYSIN.—The Council on Pharmacy and Chemistry reports that Thymophysin is stated to be a preparation of posterior pituitary and thymus, manufactured by Fritz-Pezoldt of Vienna and is marketed in this country by the American Bio-Chemical Laboratories, Inc. Because the claims for its action were inadequate, it was necessary to subject Thymophysin to examination by accurate pharmacologic methods to see whether the claims made for it are justified or whether it might not be merely a weak pituitary extract. This study was made by Prof. Erwin E. Nelson of the Department of Pharmacology at the University of Michigan. He studied the action of Thymophysin as compared with pituitary and again the action of thymus in connection with pituitary. Nelson concluded that there is no unequivocal evidence that either oxytoxic or presser activities of pituitary are altered by the simultaneous administration of thymus; that experimentally no difference could be found in the oxytoxic orpressor activities of pituitary alone as compared with pituitary plus thymus; and that Thymophysin ampules contained only about 30 per cent of the strength claimed. The Council declared Thymophysin unacceptable for inclusion in New and Nonofficial Remedies since it is an unscientific preparation marketed under false claims as to its essential action, as to its strength, and as to its safety for mother and child. (Jour. A. M. A., March 14, 1931, p. 860.)

SURGERY OF PEPTIC ULCER WITH REPORT OF FOUR RECENT CASES OF PERFORATION.

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(Presented before The El Paso County Medical Society, May, 1931.)

Surgery is indicated in only two classes of ulcer: (1) those in which the patient has been treated by a competent man for this work and has not obtained relief and (2) those cases in which the symptoms are manifested very suddenly and severely. The first type of case is one that manifests such symptoms as sour stomach, constipation, pain after eating, occasional vomiting and often a history of vomiting of blood or the passage of blood in the stools. These cases I feel belong strictly to the internist or specialist. They usually do well, many of them recovering and putting on a great deal of weight. A few others, after all methods of treatment have failed, must resort to surgery. I do not believe we should do a gastro-enterostomy when it is possible to avoid it. The other class of cases are those that perforate. I don't know what the experience of others has been, but our observation is these cases have had little or no trouble previously, none of the classical symptoms, in fact, which we have with the so-called medical ulcer. These patients as a rule have suffered but little with their stomachs and have rarely had any medical attention. The perforation usually comes on when the patient is walking the streets or is at his daily vocation. He is struck with a horrible pain in region of the stomach, as sudden as a rifle shot. His temperature drops below normal, pulse increases in frequency, the abdomen becomes rigid as a board. The diagnosis is usually easily made. We are reporting four cases which have come under our treatment during the last few months. Please note the similarity of the symptoms, and so forth.

Our method of procedure is to cauterize the ulcer, if we find one, repair the wound and use drainage for forty-eight hours if there is a perforation; if no perforation, no drainage. If it seems necessary to do further surgery, that is, make a gastrojejunostomy or gastroduodenostomy or a resection, the method is more or less a matter of choice. I would certainly not do a resection unless we had a very large ulcer about the pylorus or a large amount of cicatricial tissue. Some men are doing a subtotal resection for small ulcers. I do not think this method can be too strongly condemned. I am opposed to a gastro-enterostomy except

in cases which have had the proper medical care for some time with all symptoms persisting, such as sour stomach, pain after eating, constipation and often hemorrhage. If the symptoms are not improved I feel we are warranted in making an exploratory operation, and if you find no trouble with the appendix, gallbladder and pancreas and no definite ulcer, under these circumstances a gastro-enterostomy is the proper procedure. Formerly a gastro-enterostomy was done even for the relief of a definite ulcer; however, today the reverse is true, the ulcer is removed and the opening not made. I like a modified Finney pyloroplasty if the ulcer is not too near the pylorus. This method was the support of Judd and Horsley. Some claim the secretion of the stomach coming in contact with the jejunum is much more liable to produce ulcer about the stoma than is a pyloroplasty. We have all seen many jejunostomies after which the patients do well for a while, increasing in weight and able to eat anything; but I have seen, in some cases, after a few years old symptoms reappear and the patients suffer as before operation. Consequently, I want to emphasize again, we do not do a gastro-enterostomy except when we feel it cannot be avoided.

We shall give you briefly the method used in these four cases, three of which were perforated from two to twenty-four hours before we saw them. The ulcers in these were all excised with cautery, opening repaired, drainage instituted on account of escaped stomach contents. The fourth patient had suffered a great deal. could eat practically nothing, vomited what he ate after taking the least food. Passed blood by stools, and so forth. We did a gastro-jejunostomy and the patient was in a very weakened condition, consequently we did not interfere with the ulcer. He did well for a while but when his diet was increased the ulcer perforated and we had to do an immediate operation. We excised the ulcer with cautery and put in drainage. He had a subphrenic abscess following, which ruptured through diaphragm and was drained through the bronchi. The pictures exhibited are of this case. We had no pictures of the other three before operation. All of these patients are making good recoveries.

Case I. The first case is that of a man of 31. About half an hour before admission he was suddenly seized, while walking along the street, with a violent agonizing pain in the epigastrium, which rapidly spread downward and to the right. It doubled him up and both respiration and movement aggravated the pain. His whole abdomen felt sore and tender and he vomited once. In questioning him about previous gastric symptoms, all that I could elicit was that for a period of about two weeks just before the onset of this severe pain, and for a similar period about a year previously, he had occasional gnawing pains in the epigastrium which came on three or four hours after eating. These pains had not troubled him sufficiently to make him consult a doctor.

Physical examination revealed a rather thin man who seemed to be suffering greatly. Temperature, 96.4; respiration, 26; pulse, 64. On inspection the abdomen was scaphoid and abdominal respiratory movements were practically absent. There was tenderness and board-like rigidity of the entire abdomen, though tenderness was a little more marked on the right side. No obliteration of liver dullness could be made out. There was also tenderness in the recto-vesical pouch on rectal examination. The white count was 16,000, with 84 per cent polys.

At operation a belly full of the milky fluid usually seen in perforated ulcers was found, and there was a small perforation on the anterior aspect of the duodenum just beyond the pyloric ring. The edges of this were cauterized with an electric cautery and the opening closed by a purse string and a few Lembert sutures. One cigarette drain was left in the wound. The drain was removed after 48 hours and the patient made an uneventful recovery.

Case II. The second case is that of a man of 41. His symptoms and course are so similar to those of the first case that I shall not bother you with details. His severe pain began about 3 hours before admission and he had had practically no previous gastric symptoms. The findings, treatment, and postoperative course were the same as with number one.

Case III. The third case differs a little from the previous two. He had had, for about a year and a half before the onset of his acute illness, some gastric symptoms; namely, an occasional gnawing pain in the abdomen which usually came on an hour or so after eating and was relieved by soda or food; he had also had water brash but no vomiting. The day before admission, he woke up early with a burning feeling in his throat and took some soda which relieved it slightly. About 8 a. m. he arose and drank a glass of milk. This made the pain worse and in a few minutes it was so bad that he could not stand it. He vomited once. A doctor was called who gave him morphine and he was put on the first train for El Paso. When first seen by us he said his pain was not quite so severe as it had been.

Examination revealed a man of 24, who looked acutely ill. Temperature 100, respirations 25, pulse 94. Respirations were shallow and painful. The abdomen was scaphoid. There was marked tenderness and rigidity in the upper abdomen, especially on the right. There was little or no tenderness and rigidity in the lower quadrants. The liver dulness was obliterated. There was some question about the diagnosis in this case because of the localization of the tenderness in the upper abdomen and the fact that the pain seemed to be diminishing but we finally decided that it was a perforated ulcer.

At operation it was found that most of the milky fluid had been localized to the upper abdomen by the omentum walling it off and there was a small tab of omentum glued by fibrin over the perforation on the anterior surface of the duodenum. In fact, it was only after a search of several minutes that the perforation could be found. It was a beautiful demonstration of nature's treatment for

perforated ulcers. Our treatment was the same as in the first two cases and the patient made an uneventful recovery except for a small wound abscess.

Case IV. In this case the patient had a gastric, not a duodenal, ulcer. He came to the hospital complaining of severe pain in the epigastrium of 5 days' duration which came on soon after a dietary indiscretion. The pain was severe but not constant. It was aggravated by eating. It began in the epigastrium but radiated through to his back just mesial to the inferior angle of the left scapula and usually when the pain was at its worst his heart would palpitate. He had been vomiting practically every thing he ate for 5 days. Investigation of his past revealed that his first gastric symptoms were 2 years before admission when he had pain in his epigastrium, vomited blood and passed tarry stools. After this he had been treated medically about three times but each time he would quit his diet; and it was a heavy, highly seasoned meal which brought on the present acute pain.

Examination revealed a rather poorly nourished man of 45 who looked chronically ill and seemed to be in pain. He was vomiting brownish material which contained blood and some food eaten 6 or 7 hours previously. There was tenderness over the xyphoid and high in the epigastrium, with moderate spasm over the upper right rectus.

He was kept in bed with little or nothing by mouth for a few days and then a gastro-intestinal series was taken. This showed a large deep ulcer on the lesser curvature, and gastric retention. A few days later he was operated upon. A large indurated ulcer was found on the lesser curvature of the stomach near the pylorus. Nothing was done to the ulcer; a posterior gastrojejunostomy was done. After operation the patient did very well for a few days but as soon as he began to take any amount of fluid by mouth he began to complain of his old pain and there seemed to be a fullness in the epigastrium. The evening of the tenth postoperative day his pain became terrible and he went into mild shock. Examination revealed slight distention of the upper abdomen with marked tenderness and boardlike rigidity all over. A diagnosis of perforation was made and he was operated upon immediately. The gastrojejunostomy opening had healed perfectly but there was a large perforation at the site of the ulcer on the lesser curvature. The edges of this were cauterized and the opening closed by a purse string and two layers of Lambert sutures.

Since this operation the patient has had no gastric symptoms except on occasional feeling of fullness in the epigastrium, but he developed a subphrenic abscess and this has prolonged his convalescence. Fortunately for him the abscess ruptured through a bronchus and he expectorated the pus.

DISCUSSION

DR. F. D. GARRETT: In my opinion the first three cases described show clearly that surgical treatment was indicated and the results approved, but the fourth case would have been handled a little differently by me. This case had a retention and unless such cases are followed ten days to two weeks, we do not know whether the retention amounts to anything or not. A great many of these cases, 90 per cent of them, with ulcer near the pylorus, if put on careful treatment, will lose their spasms and their retention inside of ten days and food will be going through in the normal way. The stomach must be emptied once or twice daily during that period and plenty of glucose must be given by rectum and intravenously. Besides knowing positively whether you have retention or not at the end of ten days or two weeks, the patient will be in better condition in case you have to operate. In the particular case cited, had the patient been followed for that length of time, the ulcer would very likely have healed. I have a patient I treated over a year ago, with an ulcer fully as large as those described, with every bit as much retention of food. At the end of six weeks' treatment, she was perfectly well and has continued well up to this time, and I have seen a number of similar cases.

DR. JAS. J. GORMAN: There is nothing more dramatic than the typical ruptured -ulcer, with agonizing pain which can be read in the patient's face. If he is standing, he will be doubled over; if reclining, he will have his legs drawn up. Add to this the board-like rigidity and you have the typical picture. Less dramatic, but just as dangerous, is the very small rupture with the slow leak.

As the ulcer erodes through the mucous membrane, submucosa, and muscularis, to the peritoneum, we come to a point where a sudden strain (cough, sneezing) or the presence of an acute infection ("flu," sore throat, and so forth) may bring on a rupture. The acute ulcer is seldom guilty. Chronic, untreated ulcer is the greatest offender. Reports place ruptured gastric and duodenal ulcers as responsible for some 30 per cent of sub-diaphragmatic abscesses. The internist gets some satisfaction in the fact that the great majority of ruptures are the untreated, or inadequately medically treated, cases.

The treatment is, of course, surgical and that immediately. The longer the delay, the greater the risk, and after operation there must be close medical supervision. I regard absolute rest in the early stages as essential—rest from persistalsis and rest from secretory activity. Nothing by mouth for from three to four days, depending on the condition of the patient, except that thirst may be alleviated by a drachm of water hourly, increasing as indicated. Paraffin or gum may be chewed. Fluids, however, should be given by other methods in large amounts. If the condition of the patient requires, retention enemas or the Murphy drip may be given. Treatment can be raised rather rapidly to ambulatory and this continued from two to six months or longer, depending on the case in question.

As to the method of procedure in the case operated which later ruptured, I feel that the case was surgical from the outset. The retention, to be sure, could probably have been relieved medically; however, the fact that this man was a laborer requires consideration of the economic situation, namely, to return him to work as soon as possible. In the second place, the x-ray shows his to be a perforating ulcer on the lesser curvature, the most common site of rupture; and, third, the size of the ulcer in a man of forty-two years of age must necessarily cast a suspicion of malignancy.

DR. F. P. MILLER: The type of case Dr. Ramey mentioned—the acute perforation where previously no sumptoms had been present—is fairly common for that type of duodenal and gastric ulcer, especially duodenal. I have had three cases where men have been treated over long periods of time, one who had the advantage of medical treatment in three or four medical centers. Recently I had the pleasure of reviewing an abstract on some of the literature on the medical and surgical aspects of this subject and it seems that surgery is by no means on the defensive in this treatment.

DR. W. W. WAITE: I have seen three cases,

operated in the clinic in 1927, and all seem to suffer just as much as they did before operation.

DR. GEORGE TURNER: As Dr. Gorman said, acute gastric ulcers do not perforate. Gastric ulcers will give symptoms early, will give symptoms of hyperacidity, characterized by gastric pain, and so forth, before there is any weakening in the stomach and before anything can be found with the x-ray other than a hypermotility of the stomach. This is the time to treat gastric ulcer medically and is the time to cure the patient medically. Later, after the ulcer damages the muscle layer in the stomach wall and you get an ulcer that you can see with the x-ray, which does not come until after the muscles of the stomach have been destroyed, you have that type of ulcer which is apt to perforate. As it grows larger, the question of malignancy comes into consideration. The type of duodenal ulcer that usually perforates is in the patient seized with sudden pain who will give no history of gastric distress. A lot of these cases when suffering severe pain will give negative history so far as gastric symptoms are concerned, but operation will show, or other symptoms will indicate, the true nature of the condition.

DR. CATHCART: In regard to nonmalignant gastric ulcers, I think most of these cases recover under medical treatment. If they are x-rayed every ten days and the ulcer becomes smaller and disappears at the end of thirty days, the question of malignancy is ruled out. If the ulcer is large and remains without becoming noticeably smaller after this treatment, it is probably malignant. When it comes to duodenal ulcer, the chronic ulcer—which is the type most commonly encountered—with a definite filling defect shown by the x-ray and remaining constant and irritable over a long period of time after medical treatment, may be included in the surgical division, but a great many duodenal ulcers that bleed give very little filling defects early. These practically all recover under medical treatment with very little scar formation. Where there is very definite scar formation that exists over a long period of time with irritation, I believe these cases should be classed as surgical.

DR. BAKER (closing): I think the discussion has fully covered the subject, but would like to answer Dr. Waite. I am sure that all of you are acquainted with the work done at Mt. Sinai Hospital in New York demonstrating a resection of the pylorus, leaving the duodenum intact, but closing it and the joining the stem to the ileum. If symptoms should recur, the patient would be in great deal better condition than when first operated and it would not be very difficult to perform a secondary operation.

DR. RAMEY (closing): There is one thing I want to say in reply to Dr. Waite and that is that I know of no operation on the stomach that will guarantee relief to the patient forever. He seems to think there should never be a recurrence of an ulcer. I know of no operation that can be done that will guarantee you will not have an ulcer sometime afterwards. I have seen the entire stomach removed, even for a very small ulcer, but that carries a very high mortality and I am not in favor of it at all. In regard to the case Dr. Garrett questioned, perforation was so near complete that I believed we would have to operate upon him later anyway, and we did at that. The only mistake in my opinion was that we did not resect the ulcer at the time. As to Dr. Turner's remarks, it is a fact that cases of duodenal ulcer perforate without any warning whatever.

TOXEMIAS IN THE EL PASO CITY-COUNTY PRENATAL CLINIC

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(Presented before El Paso County Medical Society, April 13, 1931.)

Toxemias of pregnancy are generally understood to be those disorders occurraing at the time of gestation and probably caused by the accumulation within the maternal blood of toxins or poisonous substances derived from the fetus or placenta which are neither neutralized by the organs of metabolism nor eliminated by the excretory organs. Many authors believe that every pregnancy is attended by an autointoxication, due to the extra load on the general metabolism and that there is not a specific toxin.

Much work has been done in the past few years to discover the underlying conditions of this toxemia, some results of which will be of great value when the syndrome is finally worked out. Before I give the results of the work in our clinic I shall endeavor to give a brief summary of the work being done and the results being obtained in some of the large clinics and hospitals.

Titus and associates[1] offer conclusive explanation for the beneficial effects of intravenous glucose injections in eclampsia. They report nineteen eclamptic cases in which the blood sugar was examined every five minutes before, during and after the attack. The results showed a characteristic sharp fall in blood sugar just preceding the convulsion. They call this a period of relative hypoglycemia, meaning by that a level of blood sugar which, in relation to the levels shortly before, is relatively hypoglycemic, while its values in actual figures may be around normal. This is said to be similar to the hypoglycemic reaction from insulin occurring at blood sugar values of more than 200 mg. They believe a glucose impoverishment within the erythrocytes is the outstanding feature of this period. They believe that hypoglycemic levels are a predominant and fairly constant feature of true pre-eclampsia, therefore, the intravenous administration of dextrose without insulin is specific treatment for eclampsia and pre-eclampsia.

T. J. Stander and Harrison of Hopkins, did not find a hypoglycemia in a study of nine eclamptics, and are not convinced that glucose is a specific. Titus, in a detailed discussion of this paper, shows that his work was repeated in only one case by Stander.

Stander, Eastman and Harrison[2], in another series of papers, discuss the acidosis of pregnancy. They speak of a compensated

alkali deficit of normal pregnancy. During normal gestation there is a reduction in total base amounting to about eight milligrams. However, in eclampsia, at the time of convulsions and coma, there is a true acidosis dut to an uncompensated alkali deficit, as demonstrated by a definite increase in the hydrogen-ion concentration. This acidosis is not considered a causal factor, but a result of the convulsion and is the cause of death if not relieved by administration of alkali. Because of this acidosis he repeats a warning against general anesthesia.

As the liver is one of the primary organs involved in these toxemias, it has also received much investigation. All the known function tests have been run, in both normal and toxic gestations. The use of liver extract in the toxemias of pregnancy is praised by Miller and Martinez, who report a series of 43 eclamptics with a mortality of 6.9 per cent. Mendenhall and Smith reported poor results. J. L. Reycraft used heparmone in fifty toxic cases and feels that it has definite value in the hepatic cases. In a few cases alarming symptoms develop from the use of heparmone.

The kidney comes in for its share of continued investigation. John C. Hirst[3] of Philadelphia has given us a very complete paper on the kidney of pregnancy. He uses only two classifications of the toxemia:

A. The acute late gestational toxemia characterized by rapid onset late in pregnancy with good dye elimination, high urinary specific gravity and complete recovery within three months.

B. Chronic late gestational toxemia featured by gradual onset, earlier in pregnancy frequently as an exacerbation of prior hypertension, often seen in multiparae, poor dye elimination, marked eye round changes, low urinary specific gravity and incomplete recovery in three months. He believes that vasodilitation and circulatory stasis of the ureter may directly or indirectly be concerned with late gestational toxemia.

Edward J. Stieglitz, of the Chicago Lying-In Hospital, reports on sixty cases of nephritis in pregnancy in his service during the last three years. He states that the term "low reserve kidney" popularized by Stander is better called the syndrome of tissue and renal fatigue in pregnancy.

A. This group is characterized by slight arterial hypertension with only moderate albuminuria and prompt recovery.

B. The eclamptic and pre-eclamptic intoxication.

C. The toxic case in which pre-existent renal and/or vascular disease has reduced the renal reserve, with exacerbation of these processes by pregnancy.

He states that the eclamptic group is probably most safely and efficiently treated by termination of the pregnancy in the pre-eclamptic phase. Pre-existent renal and vascular disease is permanently exacerbated and degenerative processes accelerated by pregancy. Prognosis is based chiefly upon promptness and degree of improvement following obstetric delivery.

Schwartz and Dieckmann of St. Louis outline their conservative treatment of eclampsia. They recognize that mild cases if treated radically will show an increase in the mortality rate, and severe cases treated conservatively will continue to show a definite mortality if early death of the fetus does not take place or delivery occur. Difficulty persists in the early decision as to the severity of the case before too much damage is done. Their treatment includes the use of intramuscular injections of magnesium sulphate, colonic irrigations, gastric lavage, leaving 60 c.c. of a saturated solution of magnesium sulphate in the stomach and most important, frequent intravenous injections of 20 per cent glucose, 500 to 1000 c.c. each.

The results show that mild cases recover under any conservative treatment, but in the severe type recovery is markedly favored by early death of the fetus or delivery. If under treatment there is no satisfactory diuresis, coma and pulse are increasing, then dedelivery must be completed in a short time without additional shock. If this cannot be done readily through the natural passages, then abdominal cesarean section under local anesthesia.

In contrast to this, DuBose[6] of Selma, Alabama, advocates cesarean section as the treatment for eclampsia. He reports 100 operations, 60 per cent of which were eclamptics with a mortality of 2 per cent, which were not a result of the operation. His cases were all unfavorable, having been sent in from the country after having convulsions and vaginal manipulations. Previous to this series he had 10 per cent mortality under the different conservative treatments.

Dorsett[7] of St. Louis using the glucose and magnesium sulphate treatment had a mortality of 7 per cent in 186 cases of eclampsia. Cesarian section was done only nine times in this series.

McConnell[7] of Louisville goes a step further and uses continuous intravenous administration of glucose. A cannula is anchored

in the vein and 200 c.c. of 10 per cent glucose given every hour. This may be continued over a period of several days if necessary.

Kellogg[9], of the Boston Lying-In, urges the the universal adoption of Stander's classification, namely:

1. Low reserve kidney.
2. Nephritis complicating pregnancy.
3. Pre-eclampsia and eclampsia.

His report covers seven years of the clinic and 1100 toxemic cases. The only improvement in results during this period had come from active interference before the convulsive stage in the toxemia patients and before there was marked kidney damage in the nephritic group. A very significant finding was a group of 225 patients seen in two pregnancies, in which only 20 per cent were normal throughout the second pregnancy. Follow up work done by other men, namely Greenhill, Gibberd and Schmeckel, in similar large series of eclampsias, showed a similar large per cent of recurrence, but somewhat less than this figure.

Robbins and associates[9], report eight cases of eclampsia treated with sodium amytal. Moore[10], adds four more cases with favorable comment. DeLee and others think the drug dangerous and find the results disappointing.

E. Borczi[11] maintains that pregnancy toxicoses are due to congenital hypo-function of the ductless glands, especially the thyroid and advises the administration of thyroid as a preventative against eclampsia.

G. Baumgart[12] prevents eclampsia by putting patients to bed and giving them a three-day hunger and thirst cure.

In five eclamptic patients a considerable amount of blood was withdrawn by F. C. Irving and J. V. Taylor[13], the plasma was removed, the corpuscles were washed and reinfused and all the patients recovered.

A very complete report of the toxemias occurring in the city of Philadelphia in a period of fifteen months has just been published by Schumann[14]. There was a total maternal mortality of 5.4 per cent in the cases under prenatal observation and 21.5 per cent of all women having convulsions or coma.

In order to have a basis of comparison for the work done in our clinic I shall give briefly the report of the Maternity Center Association of New York. This report covers eight years and the records of 4,726 women. About an equal number of women were delivered at home and in hospitals. Physicians attended about three-quarters of the cases. Bellevue Midwife Service 17 per cent and other midwives 7 per cent.

In this group of women 15 per cent showed albuminuria, with or without some other symptoms; 5 per cent had high blood pressure only; 13 per cent had some other symptoms, such as edema, elevated temperature, bleeding, etc., while 0.8 per cent had a definite toxemia. In all, about one-third of the total number of cases had one or more symptoms of abnormality at one time or another during pregnancy. There were eleven maternal deaths showing a per cent three times better than in the cases in the same district without prenatal care. There were no deaths due to eclampsia.

In contrast to this, in our clinic, a large majority of cases were delivered at home and by midwives. These midwives, with a few notable exceptions are poorly trained and their work was done under the most difficult conditions. Physicians who were not particularly interested in obstetrics were called in consultation after many mours of labor and poor management. Some of the most difficult cases were sent in to the hospital either by the clinic or the physician called. Taking all this into consideration, I believe the results of the prenatal work done in this city have been very good. These cases have practically all been followed up by the clinic nurses who made a record of the delivery and post partum condition and saw to it that they returned for their final examination. Therefore, I feel that the records and estimate of results are accurate.

Over a period of six years there were 2,839 women in the clinic, not counting those who were not pregnant, who had a gynecological condition or who were lost to the clinic. Of this number 464 had only a post partum examination, leaving 2,375 who had some degree of prenatal care. I will not go into detail as to the number of visits of each or the month at which they first came.

J. Whitridge Williams states that 50 per cent of the pregnant women show evidence of toxemia which he classifies as pathologic. In these cases 1090 women, or 46 per cent showed at least one symptom of toxemia.

Of these, by far the largest group (595 women or 25 per cent of the total) had albumen in the urine with occasional moderate or slight edema and no other symptom. The blood pressures were normal in this group. There were five still births, two prematures and six abortions. There were ten positive Wassermanns which account for a considerable number of these. Instruments were resorted to in three cases with living babies.

A blood pressure of 130 systolic or over was arbitrarily chosen as abnormal. The next group, consisting of 242 cases or 10 per cent, recorded one or more abnormal blood essure readings. Some of these pressures ran as high as 170 occasionally, but most of them varied between 135 and 150 systolic. There were very mild or no toxic symptoms and the urine was consistently negative throughout. The records show five instrumental deliveries, five still births, one premature, two abortions, one case of phlebitis and three positive Wassermanns.

You will note that I have made no classification of the cases of hyperemesis gravidarum. In the first place they should not be classified as true toxemias of pregnancy, and secondly, in our clinic this group was conspicuous by its absence. The records show only a few cases of vomiting and they could not be truly called pernicious. A patient was sent to the county hospital last week with severe vomiting of early pregnancy. She recovered in three days under intensive treatment. Evidently this complication is rare among the Mexicans.

The next classification I have made included those cases with a blood pressure of 130 systolic or more, albumen one or more times and mild or moderate toxic symptoms. In this group there were 218 women, or 9 per cent of the total cases. There was one maternal death from pneumonia on the third day post partum, four still births, two abortions, one version and two forceps deliveries, two positive Wassermanns. I should like to add that Wassermanns have not been run routinely but only on those cases where it seemed to be indicated clinically.

That leaves us with a group of 35 patients or 1.5 per cent who have severe toxemias. Of this number the pregnancy terminated in 23 cases at home and 12 in the hospital. There were 11 still births and four abortions in these 35 cases. Three abortions were spontaneous in severe chronic nephritic cases; one therapeutic abortion in a primipara with blood pressure of 230 over 140 and history of kidney disease.

There were 17 normal deliveries of which seven were in the hospital. There was one version and two instrumental deliveries in the homes with living babies. In the hospital there were five operative cases, two instrumental, two bag inductions and one craniotomy.

In these 35 women with severe toxemias there were only six, or 0.2 of one per cent who had convulsions. Of these six all but one were delivered at home and without a single fatality.

No. 1. This patient had a pressure at the last reading of 160 over 100, albumen positive once out of three examinations. Normal delivery.

No. 2. Only one visit to clinic. Blood pressure 126 over 78, urine negative. On investigation she was found to have had convulsions. Normal delivery, still birth. This was a very rapid fulminating type.

No. 4. Blood pressure 156 over 98, albumen moderate, very marked edema. Patient was sent to the hospital and labor induced with castor oil and quinine. Normal delivery, still birth. She had convulsions four hours after delivery.

No. 5. Blood pressure rose from 104 over 64 to 160 over 100, urine showed albumen at last examination. She had one convulsion. Delivered of live baby with low forceps.

No. 6. The highest blood pressure reading was 145 over 90, urine negative. Edema very severe. Five days later she had one convulsion. Normal delivery of live baby.

Out of these 35 cases there were two maternal deaths:

No. 1. Blood pressure 128 over 74, albumen heavy in each specimen, marked edema. History of two miscarriages. Wassermann negative. She had a very large baby, was in labor two days with rising temperature. Craniotomy was done followed by death from puerperal sepsis.

No. 2. Blood pressure ranged between 160 and 140 over 96. Lrine showed traces of albumen and only moderate toxic symptoms. Normal delivery of a living baby. Mother died of heart disease.

This report tends to stress the importance of prevention of toxemia. I believe there is a tendency at the present time in the average doctor's office to neglect the other important features of the prenatal case. I would like to emphasize that the routine taking of blood pressure and urine examinations does not constitute adequate prenatal care. That is only a small but important part of a careful observation of the expectant mother.

SUMMARY

During six years of this clinic there were 2375 women who received prenatal care. Of this number 46 per cent showed at least slight evidence of toxemia; 25 per cent of the total cases showed albumen in the urine with no other symptoms; 10 per cent had only a rise in blood pressure and 9 per cent a mild toxemia. There were 35 women, or 1.5 per cent with severe toxemia and in this number six cases of eclampsia, or 0.2 of one per cent. There were no maternal deaths due to the toxemia of pregnancy.

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CASE REPORT: CALCINOSIS OF THE LEGS

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(Presented before El Paso County Medical Society, May 11, 1931.)

An American woman, past 50 years, housewife, was referred to me by Dr. C. H. Mason. She was complaining of swelling and pain in the whole left leg, causing almost complete disability. This was of several weeks' duration and had grown progressively worse.

Past History: She has enjoyed good health all her life. No severe illness. No operation or injuries. A very hearty eater, especially of meats of all kinds.

Family History: Married, one child living and well.

Physical Examination: Good musculature and well nourished. No pathology about head or neck, no goiter. Skin clear. Left leg swollen and painful to touch and movement. This pain seemed to follow the superficial veins to the groin. Veins seemed small and did not fill well in dependent position. From the knees down, many small movable subcutaneous small bodies, the size of a grain of wheat and smaller, were felt. These were very tender. There was hyperesthesia in other parts, especially over the abdomen.

The anterior and posterior tibial arteries pulsated normally. The general examination showed nothing abnormal. The urine was normal; Wassermann negative; Hb., 90 per cent; blood chemistry normal, except low calcium, 7 mg. per 100 c.c. Basal metab., —17 per cent. Skin tested for food allergy revealed but little.

X-ray showed bones of good density and normal periosteum. There were dark spots corresponding to the physical findings, but they were not connected with the bone shadow. One of these bodies was excised by Dr. W. L. Brown and seemed to be without connection with the veins, arteries, or other structure than connective tissue. The bodies were hard and felt like calcium. Dr. Waite examined them and noted no abnormal tissue. Upon crushing and adding acid, there was liberation of gas and calcium carbonate was present.

A diagnosis of calcinosis was made. In a case of Wilens and Derby, (American Journal Dis. Children, Jan. 1926) that of a child of 5 years, the pathology was studied postmortem. Blood vessel changes were marked. The endothelium was thickened and frequently bulged into the vessels. There was perivascular infiltration and fibrosis of the arterial walls. In the subcutaneous tissue there were changes in the fat lobules, between the fat cells, and often at the periphery just within the fat cells, lime occurred. In some cases the deposition of lime seemed to extend from the fat into the connective tissue. The connective tissue around the blood vessels and the nerves was not calcified. There was no fat necrosis. The initial lesion could not be identified.

Some cases have simulated Raynaud's disease. In our case no peripheral arterial disease was made out. Phlebitis was suspected but could not be demonstrated. Other subcutaneous nodules in the thigh were very tender but x-ray showed nothing. Cellulitis sometimes precedes calcinosis.

Cases of calcinosis are quite rare. The Mayo Clinic (Med. Clin. N. A., Jan., 1925, p. 1273) has reported 38 cases of calcinosis in the lungs. Such deposits have been found in various other organs, such as the stomach, kidneys and heart. Frost bite is quite a common cause of calcinosis about the ear and other exposed parts.

Calcium metastasis occurs in some cases of sarcoma, in multiple myeloma, scleroderma, and in hyperthyroidism and hyperparathyroidism; in myeloma, hypertrophy of parathyroid glandular tissues have been found.

Calcium in the blood is held in solution by protein especially, also by carbon dioxide. With an increase of protein in the blood, there is apt to be also an excess of calcium. This may throw some light on our case, because she was a heavy meat eater. However, at the time we saw her, the blood calcium was low. Calcium has a tendency to be deposited in the aorta, the coronaries, brain and splanchnic and renal arteries, where the carbon dioxide is low. In calcinosis the general opinion seems to be that the local condition of the tissues, in some way, by lack of protein or carbon dioxide, causes the calcium to be precipitated out of the blood locally.

The prognosis of calcinosis in children is very bad. In older people it is fairly good, because it is more local and circumscribed, but the disease grows progressively worse. In our case improvement occurred while thyroid was given but it was probably a mere coincidence. She was also on a low calcium diet and such foods as gave allergic reactions were eliminated.

Dr. W. H. Goeckerman, of the Mayo Clinic, in answer to an inquiry as to the treatment of calcinosis writes:

"This is a rare condition and very interesting scientifically but I know of very little that can be done for it. Occasionally we have excised such an area if it has been especially troublesome. I doubt very much that medical treatment will benefit such a patient."

CASE REPORT

(Cerebral Embolus Following Lynch Radical Frontal Sinus Operation, with Recovery.)

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(Presented before Staff Meeting of St. Joseph's Hospital, Phoenix, Ariz.)

I am presenting this case because to me it shows a very interesting chain of events following a radical frontal sinus operation.

A white female, age 39, with a negative family history, came to office complaining of sinus infection accompanied by pain, profuse dsicharge and nasal obstruction.

Past History was obtained from sister. As a child the patient was never well, having epileptic convulsions at the age of three, which have continued up until the past year. At the age of 14 the patient began having asthma, intermittent in character, but has never been entirely free from these attacks since the original onset.

Present Illness: About four years ago the patient began to have a marked sinus infection with complete nasal obstruction. She noticed that large tumor masses were beginning to protrude from the nose. She went to a local specialist, who for the following two years removed nasal polypi from time to time as they became large enough to be seen. After approximately two years of this procedure the patient went to Iowa City and had an intranasal ethmoidectomy performed upon the left side, which was followed in about three weeks by a radical Caldwell-Luc operation upon the corresponding antrum. For some reason or other nothing was done at this time on the right side. Patient obtained temporary relief from this procedure, but her asthma was only slightly improved and in about six months she began to have pain over the right frontal sinus and very severe attacks of asthma. She returned to Iowa City at intervals for slight operative procedure, and treatment, but was never able to obtain more than temporary relief. About this time the patient began to notice that she was having definite progressing blindness. This progressed very rapidly for a few weeks, but then began to clear up. About eight months ago, following the advice of her physician, she came to Phoenix hoping to get some relief from the dry climate. She immediately consulted a specialist, who advised a bilateral operative procedure, which the patient refused. Patient came to me about three months ago presenting the following findings: Partial intranasal ethmoidectomy had been performed on the left side, but a few polyps were seen in the region of the orbital ethmoids. The left middle turbinate had been removed and the inferior turbinate mutilated and scarred. No opening could be found into the left antrum, on which antrum the patient claims to have had a radical external operation. The entire right side was filled with polyps.

This is a very typical history, with which every oto-laryngologist is familiar. Phoenix is rapidly becoming the dumping ground for patients with extreme sinus infection, who have run the gauntlet of surgical procedures and finally, out of sheer desperation, come to Arizona after they have been advised that the hot dry climate is their only salvation. The question again comes up as to what is the correct procedure for this type of case. There is no doubt in my mind but that the condition is the end result of a definite allergy since childbirth. However, she was given the pollen and food tests in Iowa City, which were negative. We must then think of bacterial allergy and of Duke of Kansas City, to explain this phenomenon.

In the process of degeneration, with marked round cell infiltration, degeneration of the normal epithelial cells, arrest of function and casting off of the ciliated cells occur. There is perversion of the function of the few gland nests that normally exist, with polypoid formation of hyperplastic degenerative tissue. Nor can we facilitate the return to normal even by ample ventilation or drainage, or both, when the membranous lining of the cavity has been the seat of a chronic degenerative process of inflammation which has lasted for months or years. It ceases to be a mucous membrane. It is now a pathological membrane so distorted in its function as to have nothing in common with the normal.

In this state we see, day after day, the secretion we call pus, associated with polyp formation or hyperplasia. I cannot understand why we try to preserve this pathological state by methods of treatment which we term conservative, and in the next breath preach the total removal of the tonsil because the least remnant left will reproduce itself and cause both local and constitutional phenomena. I am becoming firmly convinced that once the lining membrane is degenerated to the point of chronic pus formation with polyps, or hyperplasia without pus, no amount of ventilation or drainage will bring it back to normal. In this state it becomes one of the most fertile sources for a secondary toxemia with symptoms that cover every field of medicine. The lymphatic drainage ways are free and numerous and the only way to deal with such degeneration is to replace it with normal mucous membrane (which is as yet impractical), or with scar tissue.

For these reasons, I cannot see why there should be a cry about preserving such tissue in the cavity. One makes no attempt to do it in other surgical procedures. The only difference is the locality. Certainly the function allotted to the accessory sinuses in man is not of a very high order, and living is coverted into one of comfort when a patient is relieved of this membrane by an operative procedure which has for its purpose the removal of every vestige of mucous membrane, not only from the frontal, but from the ethmoids, sphenoids and antrums as well.

This was carefully explained to the patient and she was given to understand that if she was going to obtain any relief, she would have to go through a long series of radical procedures. The right antrum was injected with lipiodol showing almost complete obliteration of the cavity by thickened polypoid membrane. Intranasal ethmoidectomy was performed, completely removing the middle turbinate and resecting the anterior wall of the sphenoid. As the sphenoid was opened, foul smelling pus was discharged, so disagreeable that the operation was suspended for a few minutes. Following this, a radical Caldwell-Luc operation was performed upon the right side. At the end of ten days the patient had made complete recovery and stated that she felt better than she had in many years and that she was absolutely free from her asthma. Pain over the left frontal area was very persistent, however, and she was very anxious to have this sinus cleared up before returning to Iowa. X-ray examination revealed a very large cloudy left sinus. The right frontal was very small and apparently not involved. April 28, a Lynch radical frontal was performed. The frontal sinus was found to be completely filled with pus and a few large polyps were found around

(Continued to page 334)

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TUBERCULOSIS AND ATELECTASIS

Our special tuberculosis number this month presents four papers of superior quality. Dr. Flinn has written on the old and often carefessly handled principle of rest in a way which should make it stick in our minds. The technic of examination for intestinal tuberculosis has been masterfully presented by Dr. Davis. Dr. Fales has written a good complement for Dr. Flinn's paper in giving the criteria of arrest in tuberculosis. The paper which is most likely to miss its mark is the one on atelectasis by Dr. Gatterdam, because the ideas advanced are not yet generally recognized. It is, therefore, chosen for some extended and supplementary remarks, aimed at reinforcing the conclusions advanced by the author.

Atelectasis has been a frequent subject in medical writings, particularly since 1925, but sparse indeed are the references to the condition as an accompaniment of tuberculosis. Sewall, in 1921, wrote on Pulmonary Atelectasis as a Source of Confusion in Physical Examination of the Chest.

He stated that atelectasis is probably found in every healthy lung suffering restriction of activity, and must be borne in mind in every physical examination of the chest. The term means "imperfect extension" which is a correct rendering of the etymology, since atelectasis does not necessarily predicate a complete collapse and airless condition of the pulmonary alveoli, but any reduction in volume of air below their normal state. Three stages of atelectasis should be considered. The first is physiological and temporary and occurs in any portion of the lung where inspiratory movement is deficient. The second stage represents a more of less complete collapse of the air cells within a definite area; this condition comes almost daily under the observation of the clinician, but its conscious recognition is probably rare. The third stage represents a complete collapse of air cells in a considerable area of lung, closely simulating the condition of consolidation. For a variable period after a portion of lung has been rendered airless, it may be reexpanded, but if collapse continues, the lung tissue atrophies and the area becomes a solid mass.

In spite of Sewell's adjuration of ten years ago to "think atelectasis," clinicians have until recently, given little thought to the possibility of this condition. The importance and timeliness of Dr. Gatterdam's paper cannot, therefore, be overstated. Below, we briefly review some of the important papers of the past two years on this subject:

Lee and Tucker (Atlantic Med. Jour., Feb., 1928) writing on Postoperative Pulmonary Atelectasis, quote Mastics to the effect that "pulmonary atelectasis, partial or massive, is a common post-operative pulmonary complication, and that no doubt the the majority of the conditions which have been regarded as aspiration or postoperative pneumonia, bronchitis, bronchial pneumonia, have been cases of atelectasis. Mastics suggests four clinical types; (1) the fulminant or massive, (2) the moderate, (3) the late or mild, (4) the transient type. Lee and Tucker suggest an anatomical classification into, massive, lobar, and lobular. The first, or massive, is the one usually referred to in the current literature; the second is found more frequently in medical or roentgenologic literature than the surgical; the third type has been previously described as partial atelectasis, and is the same as the evanescent type of Mastics. Following this is a very excellent description of some experimental work.

Elkin, (Sou. Med. Jour., April, 1929) gives a very excellent historical summary of atelectasis. The name was suggested by Jorg in 1832 to denote a failure of expansion in the newborn. Lichtheim, in 1878, showed that the residual air would be absorbed if the bronchus were blocked. The observations of Wm. Pasteur, beginning in 1880, mark the beginning of the modern conception of this condition; in 1910 he called attention to the fact that lung col-

lapse frequently followed operations, but held to the theory of diaphragmatic paralysis and failure of inspiratory power. In 1925, Scott and Churchill, advanced the idea of combined bronchial obstruction and weakened respiratory force. Since that time there have been numerous articles on all phases of the subject.

Cocke, (Sou. Med. Jour., March, 1929). Though no uniform classification has been adopted, he suggests the following rough grouping: (1) Pasteur's post-diphtheritic diaphragmatic paralysis. (2) Postoperative massive atelectasis, and that following chest wounds, whether penetrating or not. (3) Those due to obstruction (tuberculomas, neoplasms, foreign bodies, or mucus). (4) Those due to infections (diaphragmatic pleurisy, pneumonia, purulent bronchitis, tuberculosis. (5) Congenital.

Bowen, (Am. Jour. of Roent., February, 1929) gives as the outstanding figures in the study of atelectasis,—Sir William T. Gairdner, Dr. William Pasteur and Dr. Henry Sewall. Gairdner, when a youth of 26 and pathologist in the University of Edinburgh, wrote an extended treatise on lung collapse, and much therein remains unimproved to this day. Pasteur in 1890 published his paper on respiratory paralysis after diphtheria, and followed this at intervals with several papers on lung collapse, and to his final contribution in 1914 little has been added in point of description and diagnosis. Sewall's contribution owes its importance to the fact that he carried his studies along physiological lines, following Gairdner. Willshire and Lichtheim, and related it particularly to physical examination of the chest with regard to pulmonary tuberculosis. In this field, Sewall's contribution, until recently has stood almost alone. Bowen's paper is a very complete study of massive collapse and has an appended bibliography of over two hundred references.

Bowen has a shorter article in the Penn. Med. Jour. of Feb., 1930, on the hazard of atelectasis in diagnosing pneumonia: he closes with the statement that "the patient who, in his first hundred postoperative hours, develops a lung complication does not, in the very greatest probability, have postoperative pneumonia. The physician should think of collapse."

Chas. H. Cocke (N.E.J., of M., Oct. 31, 1929), discusses Massive Atelectasis. He states that his conception of the condition is that of development either suddenly or more chronically of airlessness in a lung, or portion of a lung, previously well aerated. He does not think that bronchial obstruction is always the cause, citing instances from reported cases where no obstruction could be found. He comments on the very few reports of atelectasis as complication in non-surgical conditions, but cites reports of cases in this group by Packard, Norris and Landis, and others. The difference between the mechanism of atelectasis and that of pneumothorax is discussed and since the term "collapse" has become associated with spontaneous and therapeutic pneumothorax, it is confusing to use the same term in describing atelectasis. He cites the case reports of Mohler and says "he likewise remarks on the paucity of case reports of its actual occurrence in medical conditions." He states again "Wilson, J. L., describes a case of hemoptysis in tuberculosis followed by massive atelectasis with recovery of the clot and reinflation of the lung after removal of the bronchial cast causing obstruction,—certainly a rare observation." He continues, "While this brief discussion deals primarily with the condition as massive atelectasis, undoubtedly varying degrees of the conditions exist from lobular, through lobar, to complete atelectasis of an entire lung . . . I agree with Miller, J. A., who, in his discussion of Packard's paper, said that in the past we must have classed as massive fibrosis of the lung and pleura conditions which are today undoubtedly suggestive of massive atelectasis." Cocke does not believe that bronchial obstruction must necessarily be present to produce atelectasis. A paralyzed diaphragm will produce it.

Colorado Medicine of November, 1930, has a symposium of six articles on atelectasis.

Sewall discussed the physiology of the process, after a brief historical survey. He calls attention to the fact that the terminal bronchioles have lumina of only 0.18 mm. (1/25 of an inch), and these open into air sacs the smallest of which are one mm. (1/25 of an inch) in diameter, so that it would almost seem that atelectasis would be the habitual thing in the resting lung. Furthermore, according to Sir Arthur Keith, a man of sedentary occupation does not use more than one-tenth of the lung tissue available for respiration; also that of the five areas on the surface of the human lung, three are in contact with stationary structures and therefore cannot be expanded. The non-moving lobule represents almost solid tissue speckled inside with microscopic bubbles of air. It may be concluded, therefore, that near atelectasis is a normal condition of lung tissue. Atelectasis is doubtless a concomitant of well night every pulmonary trouble. Not all parts of the lung are equally susceptible of atelectatic collapse; when involved in atelectasis, the outer zone to a depth of one to one and a half inches is where the consolidation is most complete. With the advent of disease, as in a lobular pneumonia, or parenchymatous tuberculosis, there is reason to suspect that local atelectasis is a constantly concomitant condition, and perhaps is a defense reaction attempting to immobilize a tissue in which motion would exaggerate the inflammatory disturbance.

Johnson discussed the pathology of atelectasis and its relation to pulmonary infection. He calls attention to the frequent observation at autopsy of small patches of collapse, usually lobular in extent and generally ascribed to bronchial obstruction. He states that massive collapse is due to a combination of bronchial obstruction and diminished force of respiration. Many factors may combine to weaken the respiratory efforts, such as pain after abdominal operations, unfavorable posture, tight dressings, abolition of cough reflex, mechanical interference with diaphragm, and many others.

Allen gives a very excellent discussion of the x-ray diagnosis. The behavior of the diaphragm must be studied following operation, if atelectasis is suspected. If there is a definite ascent of the diaphragm on one side, it will indicate actual or impending atelectasis. The x-ray signs of atelectasis embrace the displacement of heart, mediastinum and sometimes tracheal shadows toward the affected side, high diaphragm on the affected side, decreased radiolucence of the atelectasic area, increased radiolucence on the opposite side, narrowing of rib spaces, affected lung curves toward the spine and opposite lung curves away from spine, heart moves toward affected side on inspiration, restricted motion of diaphragm on affected side. From the x-ray standpoint only two main forms of atelectasis are recognized, the massive and the localized. Atelectasis is considered massive when the area involved is large enough to require shifting of mediastinal structure or ascent of diaphragm, or both, to compensate for the air absorbed; that is the type which produces the signs enumerated

above. The localized type does not have clear cut x-ray signs and requires close cooperation between members of the medical team to produce accurate diagnoses. Early bronchopneumonia and multiple areas of localized atelectasis cannot be differentiated on x-ray appearances, but if the internist reports toxic signs and laboratory evidence of infection, the roentgenologist must report in terms of bronchopneumonia. If exactly the same appearance is present without clinical evidence of infection, localized or lobular atelectasis is the proper report. Where there are large patches of relatively great density, which undergo rapid changes, they are probably atelectasis. If they persist, with slower changes in their character, they must bring to mind in postoperative cases, such conditions as infarct, embolism, abscess and bronchopneumonia.

Sante has written extensively on massive atelectasis, and has summarized his observations in his monograph on "The Chest" published as Vol. XI in the Annals of Roentgenology. He quotes Bradford's definition of clinical atelectasis, "an unusual condition in which the lung, without the presence of any gross lesion such as bronchial obstruction, pleural effusion, etc., interfering with the free entry of air, becomes airless to a greater or less degree." Sante states that "it seems most logical to suppose from the available evidence that massive atelectatic collapse of the lung results from a simultaneous inhibition of the cough reflex by some toxic or reflex stimulus in association with an impairment of the respiratory function, either immobilization of the respiratory muscle from a defense reaction or paralysis from toxic neuritis. This in turn permits the accumulation of secretions, blocking the bronchi which results in atelectasis."

Jacobaeus, in a very extensive study (Acta Radiologica, 11:547) considers atelectasis in connection with several types of lung disease, including tuberculosis, but gives this latter condition a very minor role in conjunction with atelectasis.

Simultaneously with the presentation of Dr. Gatterdam's study and conclusions, there appeared in the May, 1931, issue of The American Review of Tuberculosis, four articles on atelectasis in pulmonary tuberculosis. Hennell, writing from the New York Post-Graduate Medical School and Hospital, comments on the influence of atelectasis in the development of chronic fibroid tuberculosis of the lungs, especially the unilateral cases, concluding that atelectasis is a defense mechanism leading to healing of the diseased lung. Lloyd writes on chronic pulmonary atelectasis without bronchial obstruction, in which the onset is slow. He gives many illustrations from the Pediatrics Department of the Sea View Hospital, New York. Korol, from U. S. Veterans' Hospital, Omaha, Neb., writes on Atelectasis in Pulmonary Tuberculosis, stating that the lesion is of frequent occurrence in tuberculosis and is the cause of many physical and x-ray signs which are being attributed to the tuberculous lesions. His conclusions are not essentially different from those of Dr. Gatterdam. Glenn, of the Missouri State Sanatorium, wrote on Massive Atelectasis in Pulmonary Tuberculosis and Its Treatment by Artificial Pneumothorax; his seven cases were all in the lower left lobe, and his conclusions are also similar to those of Dr. Gatterdam.

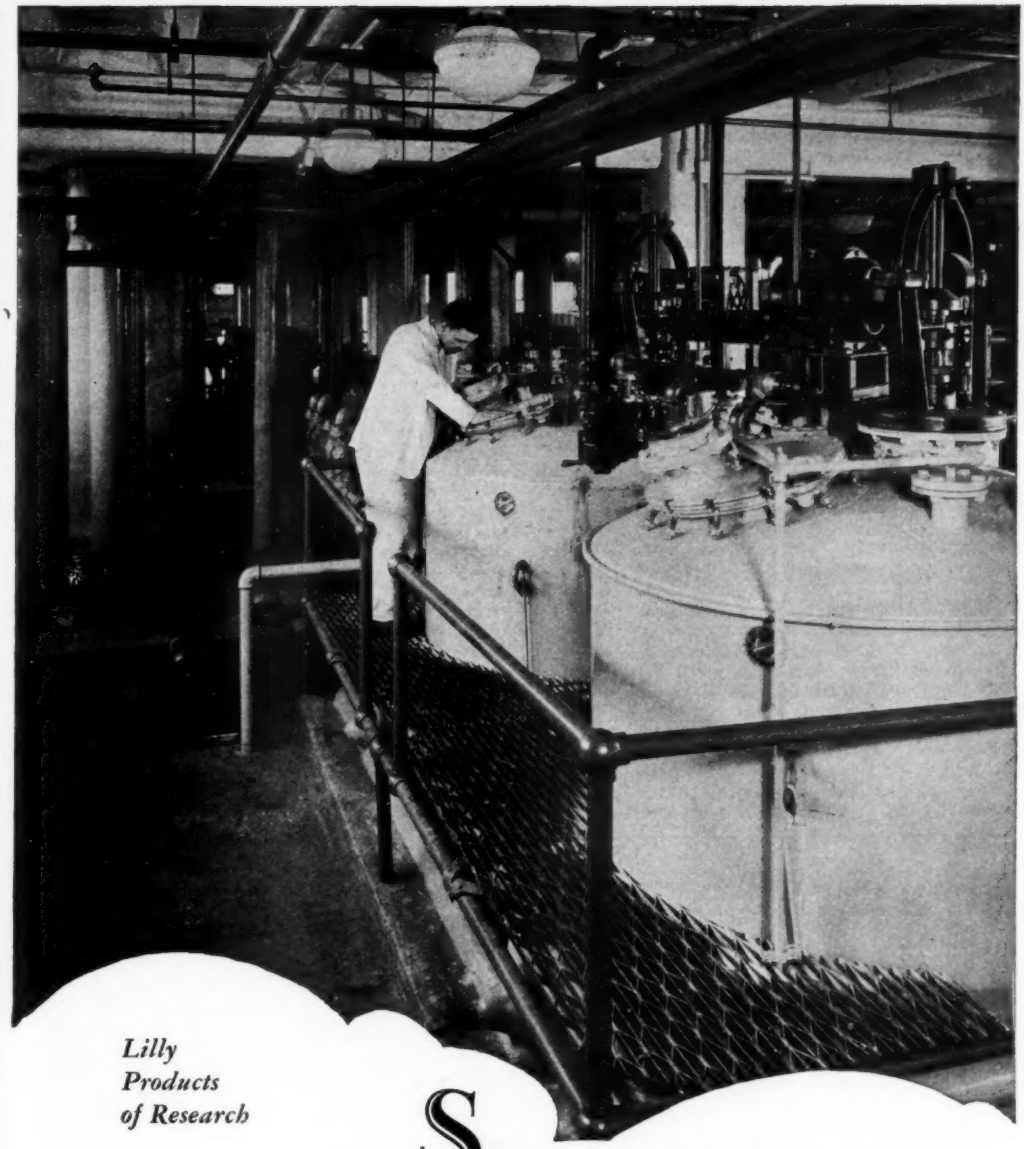
It would seem that the statements and conclusions of Henry Sewell, made ten years ago, are finally bearing fruit in clinical observations. We are entering another of those astonishing phases in medical progress, following a time when a few men, too far-seeing for their generation, drew conclusions and made predictions which have required the lapse of a decade before the main body of clinicians can appreciate their significance or importance. We will now expect atelectasis to take its place in the minds of all clinicians as a major complication in tuberculosis, or rather as a natural and inevitable part of the pathology of that disease.

CASE REPORT

(Continued from page 331)

the opening of the frontal sinus. The entire floor of the frontal, after the technic of Lynch, was removed and all the orbital ethmoid cells broken down. The brain plate was followed posteriorly and the remainder of the posterior ethmoid cells removed. The sphenoid was entered and its anterior wall dissected away. This sphenoid was also filled with a foul smelling pus. The patient was returned to her room in apparently good condition. The first day of her convelescence was rather uneventful with the exception that she complained of considerable frontal headache. About ten o'clock in the evening I saw the patient and she said that she was feeling very much better with the exception that she was having some pain in her left eye. The next morning I was called to the hospital about seven o'clock with the report that the patient had fallen out of bed and was extremely ill. Upon arriving at the hospital I found that Dr. Craig had already given the patient intravenous adrenalin. The patient was in extreme shock and looked as if she might die. The pulse was not perceptible and she was having Chayne-Stokes respiration. I remained with the patient throughout the day, administering supportive medication as her condition warranted and in the meanwhile administering intravenous glucose. After the first 1000 c.c. of glucose the patient made a very decided change for the better. Dr. Hamer saw the patient in consultation about this time and was under the impression that the patient had had a cerebral embolus, but he was unable to rule out the possibility of a cerebral hemorrhage. During the next 24 hours, the patient was apparently very much better, but then began to complain of extreme pain in her chest. Dr. Howell Randolph was asked to go over her chest but was unable to detect any pathology in the chest at that time. The next day I was called to the hospital by the special nurse who informed me over the telephone that the patient was crying bitterly with pain in the abdomen. I suspected that the patient had had a mesenteric embolus, but after about five hours the pain passed away and the patient was very comfortable. All during this time she continued to run a very septic temperature.

At the end of two weeks she was determined to go home and was discharged with a special nurse. After the third day following the removal from the hospital, I was called to the patient's home about 4 o'clock in the morning with a report that the patient had again lapsed into unconsciousness. When I arrived I found her in semi-comatose condition, but unable to speak or swallow. She could be aroused very slightly and apparently understood what was said to her but could not answer. Her condition progressed very rapidly for the worse and she passed into a deep coma. A spinal puncture was done at this time and a very hazy spinal fluid was drawn which revealed a cell count of 30,000. No organisms were found upon culture or smear.



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I again told the sister that she was going to die. During the next 24 hours the spinal puncture was done every six hours but the character of the spinal fluid did not seem to change. Upon visiting the patient the following day, much to my surprise I found her perfectly rational and sitting up in bed. Her convalescence from this time has been uneventful and I see the patient every day in the office preparatory to her returning to Iowa. She is having no headaches and is absolutely free from asthma.

Cerebral emoblus is the only thing that explains to my mind this entire chain of events. Following the embolus an infarction occurred after which the infarcted area necrosed and then ruptured through the cortex. At this time the patient suffered a marked meningeal irritation and the high spinal fluid count was due to the emptying of the necrosed area into the spinal fluid. This was evidently not an infectious thrombosis, as the patient did not develop an infectious meningitis.

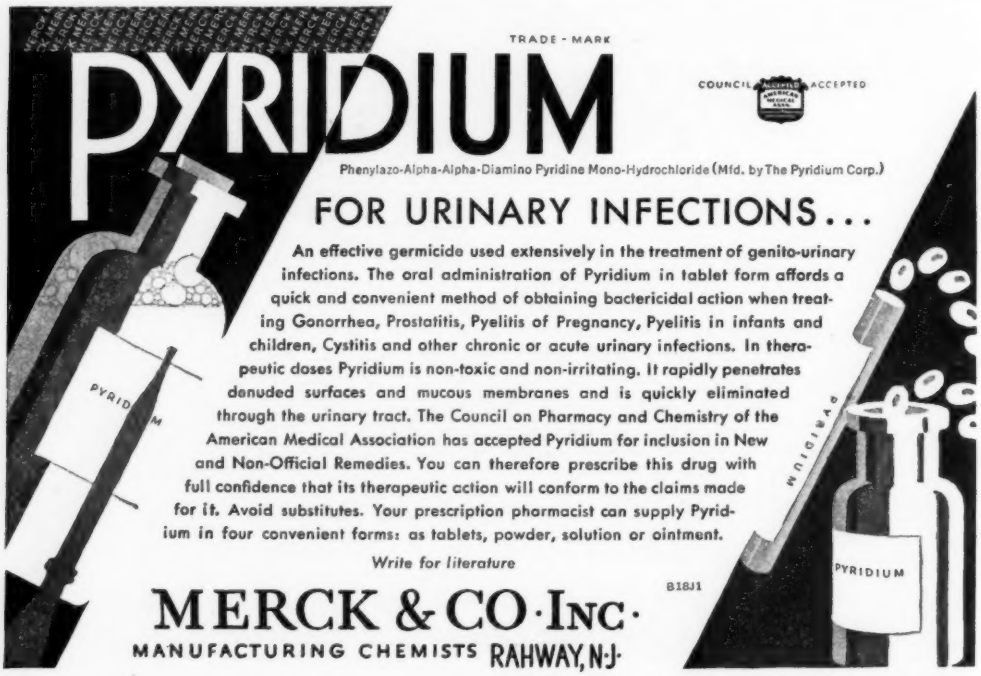
I looked up the cases of fatal postoperative embolism since the opening of the Mayo's Clinic in 1889. The total number of deaths in the hospital for the period under consideration, from all causes, was 864, of which the fatalities from embolism were approximately 5 per cent. The mortality from embolism, based on 63,573 operations, is 0.07 per cent, or 1 death in every 1,352 operations. Forty-one of the patients had been from two to thirty-five years subject to some form of severe chronic debilitating disease. Nineteen of the fatalities occurred within the first week after operation. Twenty-one within the second week, 4 in the third week and one each on the twenty-sixth, the thirtieth, and the sixty-fourth days, respectively. In 36 of the cases the embolism was pulmonary, in 10 cerebral, and in one coronary. In 28 of the 41 cases posted, the originating thrombus was found in the field of operation or the femoral vein.

DISCUSSION BY DR. W. A. SCHWARTZ

The value of climate cannot be accurately determined, but that it is of some value must be admitted. In a recent discussion of Dr. Coates of Philadelphia and Dr. Barnhill of Indianapolis both recommended Southern California and Southern Florida, and the reason was that the climate was more uniform than in other parts of the United States. Last winter an article appeared in Hygeia showing that the change in the morning and evening temperature has a stimulating effect which we do not get from a uniform temperature. If there is any value at all in climate I would think that a dry climate would be of benefit.

This patient did not derive much benefit from the climate. Radical surgery was necessary first to remove the large amount of diseased tissue. Surgery on the sinuses has not always given the result that is expected. Some patients have been made worse by surgery, but this does not mean that we should do more surgery, or less surgery. The indications for surgery should be definite and the type of operation carefully selected. The radical removal is not necessary in every thickened mucous membrane, However, when the membrane has become diseased to the extent that it has lost all normal function, complete removal is the only means to effect a permanent cure.

Surgery of the sinuses cannot be compared to surgery of the gall-bladder or tonsils. It is not always possible to remove all the diseased tissue in the sinuses, particularly in the ethmoids. The entire sinus cannot be removed, only diseased tissue as far as possible is removed. Such patients are expected to have colds and will have to withstand a re-infection. For that reason permanent and good ventilation is essential. The frontal sinus would probably not become involved if it were not for disease of the ethmoids first and blocking of the naso-frontal duct. After the frontal sinus be-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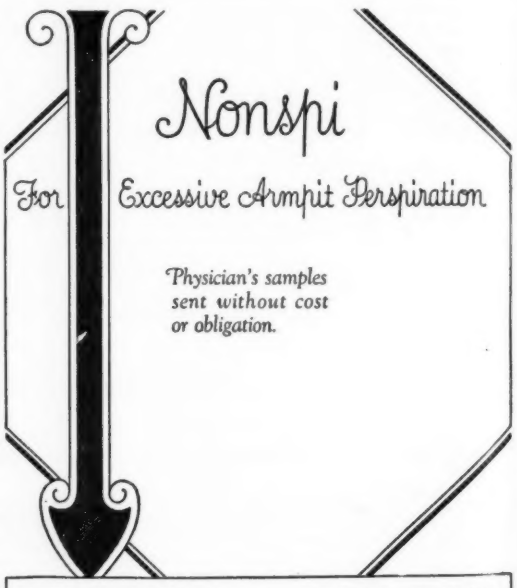
BOVININE AND NEOBOVININE.—The Council on Phermacy and Chemistry reports that Bovinine and Neobovinine are marketed by the Bovinine Company of New York and Chicago. In 1909 the Council reported that while Bovinine was advertised as a "condensed beef juice prepared by a cold process" it was not meat juice but a mixture of alcohol, glycerin, added sodium chloride, and apparently some form of defibrinated blood and that the product was marketed with unwarranted claims. In 1914 the Council reported that the unwarranted claims were for the greater part, still made, and also that the conclusions of the first report were fully warranted by the experimental evidence. The Council has not given consideration to the product since the report in 1914 was issued. Attention having again been called to Bovinine through the advertising for a similar one, "Neobovinine" which is marketed with it, a referee of the Council reported that for Bovinine the situation remained about the same as when the product was last considered. Neobovinine 20 is stated to be "essentially BOVININE (Plain) in which is incorporated the active principle of fresh whole liver." Certainly the combination of Bovinine with a liver extract of undeclared character must be held an unscientific mixture. The case histories are entirely unsatisfactory and report conditions in which it is known that there could not possibly be the results reported. The cases of pernicious anemia are not reportde with sufficient data on which to base an opinion. The Council reaffirmed the rejection of Bovinine and declared Neobovinine unacceptable because the information in regard to its composition is inadequate, because the therapeutic claims advanced for it are unwarrranted, and because the combination of "Bovinine" with a liver extract of undeclared character is unscientific. (Jour. A. M. A., March 14, 1931, p. 860.)

THE CONTROL OF NARCOTIC ADDICTION. —Narcotic addiction is again the focus of an extraordinary amount of attention. The results of investigations concerning the subject are being made public. The taking of narcotics is usually an attempt on the part of someone with little will power to achieve a state of euphoria or artificial happiness. The addict finds withdrawal accompanied by exceedingly unpleasant symptoms and tends to succumb again. Apparently it is possible for an experienced physician to carry an addict through the withdrawal period with relatively little distress. The importance of narcotic preparations should not be under-estimated. Nevertheless there exist in medicine innumerable occasions when one or more of the relatively non-habit-forming sedatives, narcotics, hynotics and analgesics may be employed to substitute for an opium derivative. A series of articles is to be published in an effort to indicate the relatively few instances demanding the administration of opium or cocaine derivatives and the many available substitutes. It is estimated that under twenty-five per cent of narcotic addiction results from prescriptions by physicians in cases in which such prescribing may not have been absolutely necessary. The physician must try to limit his prescribing of narcotics absolutely to those situations where they are indispensable. The medical use of narcotics in the United States appears to surpass that of any other nation. Efforts are being made to limit the prescribing of narcotics to those instances where their use is indispensable. (Jour. A. M. A., March 14, 1931, p. 862.)

MEDICAL ECONOMICS AND MEDICAL BUSINESS.—For some time physicians have been receivnig regularly and complimentary a publication known as "Medical Economics: the Business Magazine of the Medical Profession." The contents of this periodical are devoted largely to the problem of making money out of medical practice. It is apparently little if at all concerned with medical ethics or medical ideals. The vast majority of its space



is devoted to the advertisements of products of many manufacturers whose preparations could not possibly be passed by the Council on Pharmacy and Chemistry. Even those manufacturers who cooperate largely with the Council, find in this alleged medical publication an outlet for the announcements of their products that the Council will not accept. (Jour. A. M. A., April 25, 1931, p. 1404.)

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the bottle-fed baby exhibits symptoms indicating partial vitamin B deficiency—described by Hoobler as (1) anorexia (2) loss of weight (3) spasticity of arms and legs 4) restlessness, fretfulness (5) pallor, low hemoglobin, etc. —

Dextri-Maltose with Vitamin B may be used in adequate amounts (up to 71 Chick-Roscoe units) without causing digestive disturbance. This ethically advertised product derives its vitamin B complex from an extract of wheat germ rich in B and brewers yeast rich in G. Physicians who have attempted to make vitamin B additions to the infant's formula but who have been obliged to abandon same due to diarrheas or other unfortunate nutritional upsets, will welcome Mead's Dextri-Maltose with Vitamin B. This is a tested product with rich laboratory and clinical background and is made by Mead Johnson & Company, a house specializing in infant diet materials.

Not all infants require vitamin B supplements, but when the infant needs additional vitamin B, this product supplies it together with carbohydrate. In other cases, the carbohydrate of choice is Dextri-Maltose No. 1, 2 or 3.

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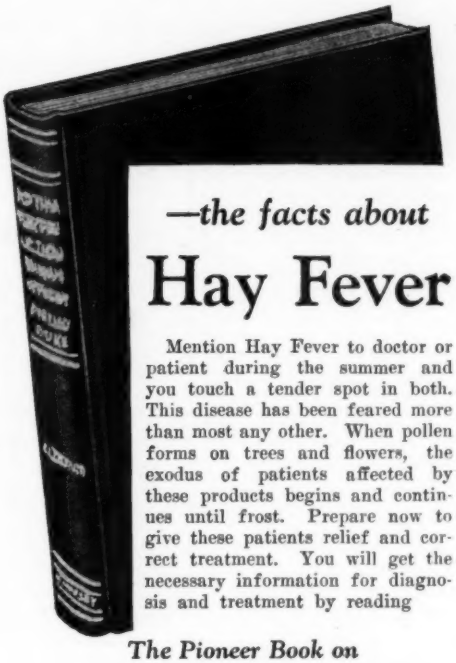

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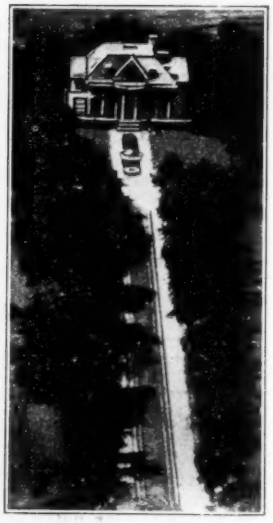
The most common causes of epileptic convulsions are toxemias, infections, tumors of the cerebrum, disturbance of the cerebrospinal fluid, circulation, faulty metabolism and injuries to the brain. In order to determine the exact cause, it is important to use every diagnostic method, including encephalography. The latter procedure has been of great value in the differential diagnosis, especially of the focal types.

The treatment consists of removal of the cause, and the use of drugs for symptomatic relief. Among the armamentarium, are the surgical removal of tumors, encephalography for the traumatic case; fluid limitation and the ketogenic diet, as indicated and certain sedative drugs, such as the bromides.

No case with a chief complaint of convulsions should ever be treated without a complete study by an individual who understands the etiological factors. Unfortunately most studies include unnecessary procedures, which both inconvenience the patient and add to the expense of diagnosis.



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